# University of Alberta

**Kinematics of Head Movement and the Role of the Sternocleidomastoid Muscle in Simulated Low Velocity Rear-end Impacts**

by

Ivonne A. Hernández

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

in Medical Sciences - Oral Health Sciences

Edmonton, Alberta

Fall 2003

National Library
of Canada

Acquisitions and
Bibliographic Services

395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque nationale
du Canada

Acquisisitons et
services bibliographiques

395, rue Wellington
Ottawa ON K1A 0N4
Canada

*Your file Votre référence*
*ISBN: 0-612-87779-5*
*Our file Notre référence*
*ISBN: 0-612-87779-5*

The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

L'auteur conserve la propriété du droit d'auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou aturement reproduits sans son autorisation.

Canada

**University of Alberta**

**Library Release Form**

**Name of Author:** Ivonne A. Hernández

**Title of Thesis:** **Kinematics of Head Movement and the Role of the Sternocleidomastoid Muscle in Simulated Low Velocity Rear-end Impacts**

**Degree:** Master of Science

**Year this Degree Granted:** 2003

Permission is hereby granted to the University of Alberta Library to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves all other publication and other rights in association with the copyright in the thesis, and except as herein before provided, neither the thesis nor any substantial portion thereof may be printed or otherwise reproduced in any material form whatever without the writer's written permission.

Date: October 1st 2003

*You have had the good fortune to find real teachers, authentic friends, who have taught you everything you wanted to know without holding back. You have had no need to employ any tricks to* steal *their knowledge, because they led you along the easiest path, even though it had cost them a lot of hard work and suffering to discover it. Now it is your turn to do the same, with one person, and another-with everyone.*

Josemaría Escrivá

**University of Alberta**

**Faculty of Graduate Studies and Research**

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **Kinematics of Head Movement and the Role of the Sternocleidomastoid Muscle in Simulated Low Velocity Rear-end Impacts** by Ivonne A. Hernández, in partial fulfillment of the requirements for the degree of Master of Science in Medical Sciences-Oral Health Sciences.

Paul W. Major, Supervisor

Gary Faulkner

Ken Fyfe

Giseon Heo

Date: October 1, 2003

Shrawan Kumar

## Abstract

The purpose of this research study was to analyze the cervical muscle response and kinematics of head movement in simulated low velocity rear-end impacts. Effect of awareness and gender differences were also investigated. Forty subjects completed the study. The results of this investigation revealed an increased SCM EMG activity and kinematic events of the head with an increased impact magnitude. The magnitude of angular displacements reported in the present study does not support the hyperextension theory. The temporal relationship of the SCM was different whether it was related to the linear or angular acceleration of the head. Temporal and amplitude awareness of a simulated impact did not produce differences in the magnitude of muscle response or kinematics of head movement. Conclusions regarding gender differences related to timing of EMG response cannot be drawn due to large variances of the variables and the low power associated with those findings.

*I dedicate this work to my friends. Their endless support, love and care were fundamental in making my dreams come true.*

# Acknowledgement

I would like to thank my supervisor, Dr. Paul Major for his direction and wisdom and would also like to thank my committee members, Dr. Gary Faulkner, Dr. Ken Fyfe, Dr. Giseon Heo, and Dr. Shrawan Kumar for their insight and feedback throughout this project.

I would like to thank Dr. Shrawan Kumar and Yogesh Narayan for the use of their lab and technical support. I would also like to thank Isaac Ikram and Michelle Adrian for their technical support and assistance in processing of the data.

I would like to thank Joanne Lafrance and Cindy Heisler for their assistance with the administration work of this project.

I would like to thank The University of Alberta Fund for Dentistry, and R.McIntyre Fund for financing this project.

## TABLE OF CONTENTS

***Chapter One- Introduction and Literature Review***


1.1 General Introduction 2

1.2 Literature Review 4

1.2.1 Whiplash injuries 6

1.2.2 Whiplash injuries and magnitude of collisions 7

1.2.3 Whiplash injuries and symptoms 8

*1.2.3.1 Neck pain in whiplash injuries 9*

*1.2.3.2 Headaches in whiplash injuries 11*

*1.2.3.3Temporomandibular joint signs and symptoms in whiplash injuries. 12*

*1.2.3.4MRI of the temporomandibular joints in whiplash injuries.* 15

1.2.4 Effect of litigation in the persistence of whiplash Symptoms 17

1.2.5 Human occupant kinematic response to a low velocity rear-end impacts 18

1.2.6 Osteoligamentous cervical model 22

1.5.7 Correlation between in vivo and human cadaver studies 24

1.2.8 EMG studies in simulated low velocity rear-end collisions 27

1.2.9 Effect of anticipation in simulated low velocity

rear-end collisions 28

1.2.10 EMG activity in patients who have experienced whiplash injuries 30

1.5.11 Biomechanics of headache and neck pain in whiplash injuries 31

1.5.12 Zigapophyseal joints as a source of neck pain in whiplash injuries 32

1.5.13 Role of the cervical facet joint capsule in neck pain 34

1.5.14 Factors that may influence the biomechanics of whiplash injuries 35

1.3 Summary and conclusions 37

1.4 Statement of the Problem 40

1.5 Statement Objectives 43

1.6 Research Hypotheses 44

1.7 References 45

*Chapter Two- Research Paper*


| | | |
|---|---|---|
| 2.1 | Introduction | 60 |
| 2.2 | Methods and Materials | 62 |
| | 2.2.1 Experimental Set-up | 63 |
| | 2.2.2 Data Acquisition | 66 |
| | 2.2.3 Experimental Phase | 67 |
| | 2.2.4 Data Processing | 69 |
| | 2.2.5 Statistical Analysis | 70 |
| 2.3 | Results | 71 |
| | 2.3.1 Kinematics of the Torso | 71 |
| | 2.3.2 Kinematics of the Head | 74 |
| | 2.3.3 Muscle response | 76 |
| 2.4 | Discussion | 77 |
| 2.5 | Conclusions | 83 |
| 2.6 | Tables | 85 |
| 2.7 | References | 89 |

## *Chapter Three- Discussion and Recommendations*


| | | |
|---|---|---|
| 3.1 | General Discussion | 95 |
| 3.2 | Limitations | 99 |
| 3.3 | Recommendations for Future Investigations | 100 |
| 3.4 | References | 102 |

*Chapter Four-Appendix*

4.1 Ethics Approval 108

## List of Tables


| Table No | Title | Page |
|---|---|---|
| Table 2.1 | Descriptive statistics of demographic data | 85 |
| Table 2.2 | Mean of the linear chair acceleration peaks in the X, Y and Z axes | 85 |
| Table 2.3 | Mean peak of the linear torso acceleration in the X and Y axes | 86 |
| Table 2.4 | Mean peaks of the angular displacement, angular acceleration and linear acceleration of the head | 86 |
| Table 2.5 | Mean of the onset and peak times of the angular displacement, angular acceleration and linear acceleration of the head. | 87 |
| Table 2.6 | Mean of the electromyogram peaks of the right and left SCM muscles | 88 |
| Table 2.7 | Mean of the onset and peak times of the electromyogram peaks of the right and left SCM muscles | 88 |

## List of Figures


| Figure No | Title | Page |
|---|---|---|
| Figure 1.1 | Ball and chain analogy | 20 |
| Figure 1.2 | In situ cervical whiplash motion | 25 |
| Figure 1.3 | Open book motion | 26 |
| Figure 1.4 | Instantaneous Axis of Rotation | 26 |
| Figure 1.5 | Facet joint pinching mechanism | 31 |
| Figure 1.6 | Impingement of the synovial folds of the zygapophyseal joints | 33 |
| Figure 2.1 | Car seat mounted in the sliding board | 64 |
| Figure 2.2 | Multipurpose circuit board SB1 | 65 |
| Figure 2.3 | Graphic representation of the global axis | 66 |
| Figure 2.4 | Set up of a participant for an unexpected impact | 68 |
| Figure 2.5 | Comparison of video camera and accelerometer system | 72 |
| Figure 2.6a | Graphic representation of the SCM EMG activity, head Angular acceleration and angular displacement of the head upon impact | 73 |
| Figure 2.6b | Graphic representation of the SCM EMG activity, head acceleration (X and Y axes) and torso acceleration upon impact | 73 |

## List of Abbreviations and Symbols

| | |
|---|---|
| CT | Computed tomography |
| EMG | Electromyography |
| IAR | Instantaneous Axis of Rotation |
| MRI | Magnetic resonance imaging |
| ms | Miliseconds |
| MVA | motor vehicle accidents |
| SCM | Stenocleidomastoid |
| SPSS | Statistical Package for the Social Sciences for Windows software |
| TM | Temporomandibular |
| TMD | Temporomandibular Disorders |
| TMJ | Temporomandibular Joint |
| WAD | Whiplash Associated Disorders |
| $\Delta V$ | Velocity change |

# Chapter One

## Introduction

## And

## Literature Review

## 1.1 General Introduction

Whiplash injuries and motor vehicle accidents are strongly related. Rear-end impacts result in a higher frequency of whiplash injuries in comparison with other types of car accidents[1]. The biomechanics of the whiplash phenomenon in low velocity rear-end impacts[2-4], the role of cervical muscles[5-8], the most prone sites to injury[6 9-11], and the effects of litigation[12-14] have been studied in order to develop a rationale for the persistence and severity of symptoms reported by subjects who have undergone whiplash injury.

Crowe[15] introduced the term "whiplash" in 1928, but it did not appear in the medical literature until 1945[16]. Whiplash implies medico-legal connotations and invokes controversies that have mainly focused on what specifically constitutes the biomechanical process of whiplash and how the whiplash mechanism injury occurs.

In 1995, the Quebec Task Force[17], under the leadership of Spitzer, conducted an exhaustive literature review of whiplash. It reported that although there were a significant number of whiplash-related scientific articles, only a few were of a good scientific quality. The Quebec Task Force[17] defined whiplash as "an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side impact motor vehicle collisions, but also can occur during driving or other mishaps. The impact may result in bony or soft tissue injuries (whiplash injuries), which in turn may lead to a variety of clinical manifestations (whiplash associated disorders)."

Recently, Barnsley et al[18] have indicated that whiplash comprises three different aspects: "*the whiplash event,* a biomechanical process suffered by the

occupant of a vehicle that is struck by another vehicle; *the whiplash injury,* the impairment that ostensibly results from the whiplash event; and *the whiplash syndrome,* the constellation of symptoms that are attributed to the supposed whiplash injury."

Whiplash injuries are a major health problem, and they have significant economic consequences in most industrialized countries. Castro et al[7] reported that 65% of insurance claims in Germany involved low velocity impacts and that between 10 and 20 billion Deutsche Marks are paid out in Europe each year to individuals who were been involved in MVA and have experienced whiplash injuries. More than a million whiplash injuries occur each year in the United States[19]. Although not catastrophic, these injuries have significant economic impact and are estimated to cost the United States $4.5 billion annually[20]. In Canada, Spitzer et al[17] reported that approximately 5000 whiplash cases annually accounted for 20% of traffic injury claims in Quebec. Teasell and Shapiro[21] indicated that "despite the significant socio-economic impact of whiplash injuries, high quality scientific research is only beginning to emerge."

## 1.2 Literature Review

"Whiplash syndrome[18]" and "whiplash injury[18]," specifically, cervical injury, have been extensively investigated. Research on this topic has been conducted through three main types of studies: retrospective and prospective analyses, studies that use human volunteers in simulated low velocity rear-end collisions, and those that involve the use of human cadavers. In turn, data of these studies have been used to build mathematical models that simulate the biomechanics of whiplash.

Retrospective and prospective studies report the most predominant signs and symptoms of whiplash, treatment outcomes, effects of litigation, and parameters that play a role in the severity and persistence of WAD.

Human cadavers have been used to describe the kinematics events in whiplash. In addition, they have been useful to identify the sites most prone to injury, to hypothesize the mechanism of whiplash injury, and to determine the injury threshold acceleration level. The use of human cadaver specimens as cervical models, provide biofidelity: the size and structure of the components of the neck are human and, therefore, the masses are accurate[22]. The main limitation of this model is the state of the muscles; cadaveric muscles impose an artificial stiffness on the neck. However, cadavers in which the cervical muscles have been removed provide invaluable information about the kinematics of the cervical spine[23].

Studies using human volunteers[3 4 24-26] have been useful to describe head and neck kinematic response to low speed rear-end collisions, to investigate the role of cervical muscles, and to identify the factors that may influence the neck response in whiplash. The main advantage of using human volunteers over mathematical, animal

or human cadaver models is biofidelity. However, the use of human volunteers also has two limitations: ethics and apprehension. Participants can be exposed to only minimal impacts and volunteers expecting an impact may have greater anxiety than those who are involved in a real collision[22].

Human cadaver studies and studies using volunteers have confirmed the kinematic events and the severity of whiplash injuries with the concomitant use of high-speed video cameras[3 9 25], as well as imaging techniques. MRI[7], functional radiographs[27 28], and cineradiographic techniques[11] are a few examples.

Mathematical modelling of the cervical spine has the advantage of not requiring experiments on animal models, cadavers, or human volunteers to depict the normal kinematics as well as to predict cervical spine behaviour when it is exposed to abnormal loads[22]. However, validation is mandatory in this type of model[29] and no mathematical model has yet shown accuracy in the reproduction of segmental cervical movements[22].

Mathematical modelling of the TMJs invokes a scientific challenge due to the numerous muscular forces and various mandibular movements that take place in the TMJs[30-33]. TMJ articulating surfaces are irregularly shaped and they are covered with viscoelastic cartilage. The mandible acts as a deformable body. The jaw closing movements are controlled by a combined action of various masticatory muscles. Joint reaction forces and their forces also contribute to the mandibular movements[31]. Koolstra et al[32] suggested that the human masticatory system is a kinematically and mechanically undetermined system. Recently, May et al[30] built a mathematical model

using a series of mechanical assumptions. They estimated that the joint force for normal males and normal females were 260N and 172 N respectively.

Prospective and retrospective analyses, human cadaver studies and studies using human volunteers will be analyzed. Their contribution to the understanding of the clinical picture and the mechanism for whiplash injury will also be discussed in the following sections.

### 1.2.1 Whiplash injuries

Whiplash injuries represent the classic example of soft tissue injury to the neck. Soft tissue injury implies that there is an absence of bone injury, and if anything has been injured, it must be one or more muscles or ligaments of the neck[22]. Most of the time, whiplash injuries are sub-failure injuries; in other words, there is incomplete failure of the soft tissues. This fact explains why these injuries are undetectable on plain radiographs[34 35]. Furthermore, imaging methods such as CT scan or MRI do not have sufficient resolution to identify them[27 36]. Indeed, in a recent literature review, Kaiser et al[37] suggested that MRI was not recommended in assessment of whiplash injury unless signs or symptoms of cord injury or radiculopathy were present.

Cervical whiplash injury is difficult to visualize and to quantify. Clinically, a flexibility test has been proposed as a good diagnostic tool to quantify them. Dall' Alba et al[38] compared the cervical range of motion of a group of subjects who presented persistent WAD with an asymptomatic control group. They reported that there were significant differences in range of cervical movements between the groups. Ostebauer et al[39] reported similar findings.

Radiographically, the measurement of the IAR, not the measurement of cervical range of motion, has been useful to provide information about motion and therefore the abnormal quality of cervical motion. When a cervical vertebra moves from full extension to full flexion, its path appears to lie along an arc whose centre lies somewhere below the moving vertebra. This centre is called IAR. IAR can be reliably and consistently calculated within a small margin of technical error, it is independent of whether it is calculated on the basis of anteflexion or retroflexion films, and it is constant over time[40]. It has been reported that this axis shifts its location upward in crash collisions[11].

### 1.2.2 Whiplash injuries and magnitude of collisions

It seems that there is still no agreement regarding the minimum magnitude of the impact at which patients report clinical symptoms. Castro et al[7] stated that the biomechanical "limit of harmlessness" in rear-end collisions is at a $\Delta V$ between 10 and 15km/hr; morphological and anatomic signs of injury to the cervical spine cannot be demonstrated within this speed range. West et al[41] concluded that subjects can tolerate impacts up to a $\Delta V$ of 8km/hr without experiencing injuries. Szabo and Welcher[2] stated that a healthy human can withstand a rear-end impact with a $\Delta V$ of 8km/hr without sustaining significant injury, assuming the presence of a head restraint and a normal initial seating position. McConnell et al[3] reported that it would be unlikely that significant injury results at a $\Delta V$ of 9km/hr or less.

Brault et al[42] reported that discomfort might even appear at a $\Delta V$ of 4km/hr. Ferrari[43] indicated that the significant differences in their results from the other

studies might be due to the subject selection criteria, symptom amplification, and symptom attribution. He also indicated that part of these inconsistencies might have been eliminated by asking the participants to complete a daily diary describing all aches and pains for a period of time (for example 6 weeks). In addition, the authors could have used a placebo collision with a control population[44]. In fact, Castro et al[45] reported that a placebo collision might induce WAD in almost 20% of subjects, and therefore, they underscored the importance of analyzing scientific results with caution, especially if the study is conducted without a control group subjected to a placebo collision.

### 1.2.3 Whiplash injuries and symptoms

Prospective and retrospective analyses have stated that patients who have experienced whiplash injuries may report a multitude of symptoms including neck pain[46], headaches[47,48] and TMJ signs and symptoms[13 49-51]. Cognitive impairment symptoms have also been reported as a sequela of whiplash. However, a recent literature review conducted by Radanov et al[52] indicated that unfortunately, most of the studies that reported these kind of symptoms lacked an adequate sample size or included patients who experienced loss of consciousness. They highlighted the fact that loss of consciousness implies head injury and; therefore, such patients should not be diagnosed with whiplash.

Various attempts have been done to predict the progression of the whiplash symptoms. Gargan et al[53] reported a relationship between the severity of whiplash symptoms and restriction in neck movements and psychological disorders. However,

they stated that the psychological disorders occur after the physical disorder. Panjabi et al[54] stated that the severity of whiplash trauma usually does not correlate with the seriousness of the clinical symptoms. Ritcher et al[55] reported a correlation between the severity of whiplash injury and the duration of the symptoms; however, Sturzenegger et al reported the opposite[56]. Recently, Suissa et al[57] suggested that higher number of whiplash symptoms was strongly associated with longer periods of recovery from those symptoms.

*1.2..3.1 Neck pain in whiplash injuries*

Neck pain represents the most important and most common complaint of patients who have experienced whiplash injuries[58-60]. Borchgrevink et al[61] reported that 58% of a patient population, who were involved in MVA, reported chronic symptoms after neck sprain injury. Patients usually describe their neck pain as an ache or as a stiff neck[62]. Maimaris et al[63] conducted a retrospective study of 102 patients who were involved in MVA and experienced whiplash injury. It indicated that the most commonly reported symptoms were neck pain, neck stiffness, shoulder pain, and headaches. Karlsborg et al[64] conducted a prospective study to determine signs that predict clinical outcomes in patients who experienced whiplash. They indicated that neck pain was the most common complaint among participants, and all the patients reported neck pain at the initial examination. 94% of the patients had limited range of cervical motion at the initial appointment, while only 32% had restricted cervical movements after several months. In another prospective study,

Pearce[47] reported that 100% of the patients of a population who experienced whiplash injuries presented neck pain.

Kasch et al[65] investigated the relationship between neck mobility, neck pain, and headaches. They indicated that the group who experienced whiplash injury had reduced cervical motion within the first three months after cervical trauma. Their results also revealed an inverse relationship between neck pain and neck mobility. There were no significant differences in neck mobility between the control group and whiplash injury group after three months. Based on this finding, Kasch et al[65] suggested that whiplash injury did not give rise to persistent or long-term restriction of neck mobility.

Friedman and Weisberg[66] conducted a retrospective analysis of 300 patients who experienced whiplash trauma. They reported that the most common abnormal cervical findings were facet joint tenderness, painful and restricted cervical range of movement, and trapezius tenderness. In a similar type of analysis, Kolbinson et al[67] reported that 94% of patients who were involved in MVA reported neck pain whereas Magnusson[68] reported that 100% of patients experienced neck pain as a consequence of whiplash injuries.

Norris and Watt[69] reported that the prognosis of neck injury as a consequence of whiplash depended on the absence or presence of degenerative changes, radiographic abnormalities of the cervical spine, or both. Gargan et al[53] suggested that reduced cervical range of motion is a good predictor of recovery from whiplash injuries. Squires et al[60] conducted a fifteen-year follow up of soft tissue injuries of the cervical spine in patients who experienced whiplash injuries. They reported that 18%

of patients had recovered from their symptoms between 10 and 15 years after the accident, whereas 28% had deteriorated. They also reported that psychological disturbances were present in more than the 50% of the patients.

Interestingly, Steigerwald et al[70] reported that the persistence of symptoms such as neck pain, dizziness, headaches, neck pain, shoulder, and tinnitus in a trauma related TMD patient population is because of intraarticular pathology of the TMJs. 98% of the patients reported improvement of their overall pain after undergoing arthroscopy of their TMJs. The rate of improvement for the rest of the symptoms were above 90%, but neck pain was 88%.

*1.2.3.2 Headaches in whiplash injuries*

Headaches are the second most often reported symptoms of patients who have experienced whiplash injuries. Magnusson[68] reported that 97.4% of patients who experienced whiplash injuries suffered from headaches. The most common reported location for headaches is the occipital region, followed by generalized headaches, and then other locations[47 71 72]. Lord et al[73] conducted a study to determine the prevalence of "third occipital nerve headache" associated with neck pain in one hundred patients who experienced whiplash injuries. 71% of the patients complained of headaches associated with neck pain. 40% of patients reported headaches as the main complaint, whereas 31% of the patients described them as a secondary complaint. In another study[74], 37% of the patients were diagnosed with tension type headaches, 27% with migraines, 20% with cervicogenic headaches, and 18% of the patients who experienced headaches did not fulfill any diagnosis of the International Headache

Society classification. 93% of the patients reported that their headaches were associated with neck pain. Most of the patients described their headaches as a pressing or tightness frequently accompanied by a throbbing or a burning sensation.

Pearce et al[47] conducted a prospective study to analyze the relationship between headaches and whiplash injuries. They reported that 60% of the patients reported headaches as a consequence of their accidents. 83% of the patients reported that the onset of their headaches occurred within 24hrs. Most of the patients described their headaches as a generalized dull achy pain with a mixture of ache and tightness. Tension-type headaches disappeared within three weeks. 48% of the patients reported that the headaches did not prevent them from working.

#### *1.2.3.3 Temporomandibular joint signs and symptoms in whiplash injuries*

Various attempts have been made in order to determine the prevalence of TM symptoms associated with whiplash injuries. Pullinger and Seligman[75] analyzed the history of trauma in patients who experienced TMD, reporting that the history of trauma was 30% more frequent in TMD patients than in the control group. Burgess et al[50] reported that 30% of the subjects who experienced trauma to the cervical and/or facial region identified overt trauma as a precipitating factor of their facial pain and TMD. Kolbinson et al[67] clinically assessed patients with TMD that resulted from MVA; 66% of the patients reported TMD as the main complaint. Braun et al[49] conducted a cross-sectional study to determine whether TMD was associated with whiplash injuries. They reported that participants with cervical trauma had

significantly more pain associated with jaw function, limited jaw mobility, and more evidence of mild to moderate intracapsular TMD.

Kronn[76] compared symptoms resulting from cervical whiplash between forty patients who experienced this type of injury and a matched control population. She reported that TMJ pain, limitation of mouth opening, and masticatory muscle tenderness were found significantly more frequently in the patient group. However, the presence of TMJ sounds, deviation during mouth opening, and the overall presence of symptoms were not significantly different between the groups. De Boever et al[77] reported that 24.5% of their TMD patient population presented a direct link between trauma, specifically whiplash injuries, and onset of pain and dysfunction.

Burgess et al[51] assessed the potential relationship between patient self-reports of jaw posture at impact and self-reported symptoms and examination signs suggesting whiplash and TMJ injury. Their findings indicated that at the time of the examination, 82% of subjects reported facial pain, 57% noted head pain, 44% temple pain, and 47% TMJ pain. During the examination, 43% were found to have either crepitus, clicking, or popping of the TMJ, 41% of subjects had one or two masticatory muscles that were tender to palpation, and 37% had three or more muscles that were tender. The mean maximum opening was 37.52mm, and 28% of the subjects had an unassisted jaw opening lesser than 30mm. They indicated that their results suggested that TMJ or masticatory muscle injury might be associated with varying crash characteristics.

Kolbinson et al[78] analyzed the role of motor vehicle accident trauma in the incidence of signs and symptoms of TMD. They compared patients with trauma

related TMJ symptoms with non-trauma related TMJ symptoms. Posttraumatic TMD patients reported a higher rating of facial pain on initial examination. They also presented greater masticatory muscle, neck muscle, and TMJ tenderness. They were diagnosed with myofascial pain and arthragia/capsulits more frequently. Friedman and Weisberg[66] reported a retrospective analysis of 300 patients who experienced whiplash and were treated for cervical and TM symptoms. They indicated that the most frequent abnormal TM findings were masseter tenderness, TMJ range of motion abnormalities, lateral TMJ tenderness, and disc derangements. They also reported that the most frequent components of the diagnosis of TMD were myofascial trigger points, hyperactivity (spasms) of the jaw closing muscles, and TMJ synovitis.

Most of these studies concluded that a comprehensive examination of the TMJ and cervical complexes should be considered as part of the mandatory examination that a patient should receive after suffering a whiplash injury.

Probert et al[79] reported, somewhat surprisingly, that only 0.5% of an Australian population who suffered from whiplash injuries sought treatment for TMD symptoms. They also reported that whiplash was found to be the most frequent injury associated with subsequent temporomandibular pain dysfunction disorder. Recently, Kasch et al[80] conducted a prospective study and compared the development of TMD symptoms in a group of patients who experienced whiplash as a result of a collision with a group of patients who had ankle injury. Their results revealed that whiplash was not a major factor for the development of TMD. Heise et al[81] studied the incidence of associated TMD in patients who were diagnosed with whiplash injury. They reported that the incidence of TMJ pain and clicking, following whiplash injury,

was extremely low and that patients who did not have clicking on resolution of their initial pain/dysfunction subsequently did not develop this sign.

None of the studies that reported a relationship between whiplash and TMD were able to establish causation; in this regard, McKay and Christensen[82] indicated that "it is an unreliable pseudoscientific speculation to establish causation between somatic TMJ disease/dysfunction and MVA based on anamnesis and/or symptom data studies."

#### *1.2.3.4 Magnetic resonance imaging of the temporomandibular joints in whiplash trauma*

There is an agreement regarding the accuracy of MRI to predict disc position and disc form in the TMJs[83-86]. It has also been proposed that whiplash injuries are strongly related to internal derangement of the TMJs[87]; however, mixed results have been reported regarding this hypothesis. MRI is the most common imaging method used to assess whiplash injury to the TMJs[88]. Schellhas[89] investigated the correlation between clinical and radiologic findings in thirty patients who sustained injuries to the TMJs, ten of whom experienced whiplash trauma. They reported that twenty-seven asymptomatic patients, who underwent MRI or arthrography of their TMJs after injury, exhibited radiologic changes suggestive of early to advanced joint derangement. They suggested that whiplash injuries might result in soft tissue trauma and aggravate a pre-existing joint derangement.

Pressman et al[88] conducted a retrospective analysis of MRI of the TMJs with the purpose of determining the frequency of internal derangement and/or other related

abnormalities of the TMJ. They reported that 88% of the patients, who did not have history of trauma, had some type of abnormality of the TMJ. 65% of the TMJs showed the presence of effusion with 15% of these accompanied by disc displacement. Thirty-seven TMJ images were diagnosed with disc displacement: twenty-one with anterior disc displacement with reduction, six with lateral or medial displacement, and one with posterior displacement.

García et al[90] investigated the relationship between cervical whiplash and TMJ injuries with the aid of MRI. Patients included in the sample were subjects who experienced whiplash injury and were diagnosed with TMD as a consequence of the cervical trauma. Based on MRI findings, 95% of the participants had TMJ abnormalities. 72% of these patients presented disc displacement with reduction and 15% had disc displacement without reduction. 69% were diagnosed with effusion and 51% had inflammation or edema. García et al[90] suggested that there was a strong relationship between cervical whiplash and TMJ injuries. They also indicated that TMJs should be routinely imaged with magnetic resonance in those patients who have experienced whiplash injuries.

Bergman et al[91] studied the incidence of internal derangement of the TMJs following whiplash. The sample was composed of sixty patients who had undergone whiplash injury and fifty-three healthy volunteers who represented the control group. They did not find significant differences in MRI between the two groups regarding presence of disc displacement or effusion. The differences in their results from those of García et al[90] could be accounted for by the fact that in the latter study, the sample was strictly composed of subjects who underwent whiplash injuries and experienced

TM symptoms. Furthermore, García et al[90] did not include a control group, and MRI were taken up to a year after the accident.

Bertram et al[92] emphasized the importance of using MRI as a complement to the diagnosis of TMD. Their results revealed that clinical pain was not correlated to the presence of internal derangement of the TMJs. They strongly suggested that MRI was necessary to establish the presence or absence of internal derangement.

### 1.2.4 Effect of litigation status in the persistence of whiplash symptoms

Kolbinson et al[13] conducted a comprehensive literature review regarding the impact of litigation on the outcome of TMJ injury-related symptoms. They indicated that most of the studies revealed that whiplash symptoms did not subside shortly after the settlement. In addition, several studies stated that an early settlement of claims might improve the prognosis of the WAD. They suggested that whiplash injuries imply confounding biologic and psychologic factors, and therefore, based on their literature review, Kolbinson et al[13] stated there was insufficient evidence to support a purely organic or psychologic origin for whiplash injuries. In a later study, Steed and Wexler[93] suggested that TMD, whether traumatic or non-traumatic, preceded psychosocial problems, rather than psychosocial problems leading to TMD.

Kolbinson et al[12] conducted a telephonic survey to investigate the relationship between the persistence of TMD symptoms and litigation status. Despite of the limitations of their pilot study, they concluded that legal claims do not have a significant impact in jaw pain or dysfunction levels. Kolbinson et al[94] drew similar conclusions. They suggested that litigation had little or no effect on the type of TMD

treatments and TM treatment outcomes. However, Cassidy et al[95], also using a survey, reported a strong and consistent association between the settling of litigation and indicators of recovery from whiplash injuries. They indicated that lower pain levels, a higher level of physical functioning, and the absence of depression were strongly associated with a shorter time to claim closure.

### 1.2.5 Human occupant kinematic response to low velocity rear-end impacts

Severy et al[96] were one of the pioneers in this type of study in 1955. They conducted simulated impacts using volunteers and dummies at $\Delta V$ ranging from 7 to 20 km/hr. Some of their conclusions are still valid today. For example, they reported that the whiplash injury could be significantly affected by the $\Delta V$ at which the impact occurs, the type of car (mass and collapse characteristics), seat back (height and strength), human occupant response, and preparedness of the occupant to the impending collision. They also reported that neck injury is likely located in the cervical soft tissue.

Other studies, using a modified and improved experimental design, have described the human kinematics in different phases. Each of these phases has specific features and characteristics. The study of McConnell et al[3] will be described and compared with similar studies.

McConnell et al[3] conducted fourteen simulated rear-end impacts with a $\Delta V$ range of 5.8-10.9km/hr. The resulting head, neck, and torso kinematics from a total of eighteen subjects were recorded using a variety of improved electronic and high-

speed film-based data collection methods. They described the kinematic events in the following phases:

Phase 1-Initial Response: 0-100ms. Within the first 50 to 60ms after impact, the body remained motionless. Between 60 and 80ms the pelvis pulled the upper body forward from below, while the lower portion of the trunk intersected the backward decline of the seat back. The mid back then began to move forward along with the seat back, and as a consequence, the thoracic curve became straightened out. By 80ms, the subject's T-1 had moved forward about 2.54cm.

During this period, the head stayed completely motionless, even though the base of the neck had also moved forward along with T1. The normal neck muscle tone that keeps the head erect began to exert a forward pull to the top of the neck, and by 80ms and onward, the forward accelerative forces at the top of the neck built rapidly, and by 100ms, the top of the neck had just begun to move. By this time also, T1 and the base of the neck moved forward another 1-2cm.

Phase 2-Principal Forward acceleration: 100-200ms. In this phase, the seat back reached its maximum rearward deflection (10-14 degrees). The neck still appeared oriented almost vertically at this point. Between about 110 and 170ms the head rotated about 10-15 degrees and then started to translate forward while the base of the neck continued to move forward. Around 180-200ms, the head reached its maximum rearward rotation and the neck reached its maximum extension of 18-51 degrees. At 180-200ms the seat back deflected, and the torso and T1 angle had decreased to about 5-6 degrees. The results revealed that all subjects had less neck extension than their maximum voluntary neck extension as recorded prior to testing by about 10-40

degrees. The authors stated that the ball and chain analogy represents a good example to explain the self-limitation of the head's rearward rotation and neck extension. As the base of the neck continued to be accelerated forward, there was traction at the top of the neck and at the point of the neck's attachment to the head (Figure 1.1). The further the head rotated the occipital condyles forward and up, the greater the resistance from C-1, which was being pulled down and forward.

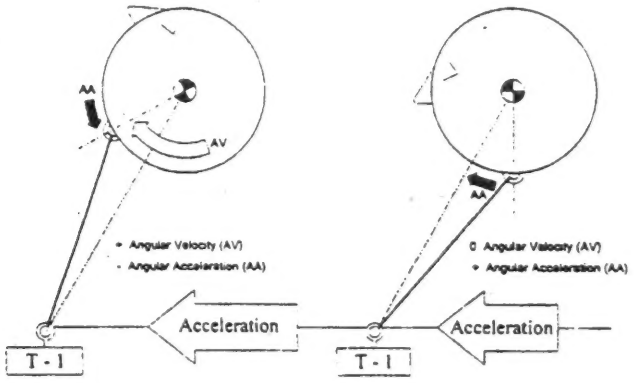


Figure 1.1: Ball and Chain analogy. Extracted from Human head and neck kinematics after low velocity rear-end impacts-understanding whiplash. McConnell et al 1995[3].

Phase 3-Head Overspeed/Torso recovery. 200-300ms: In this period, the head achieved a somewhat greater velocity than the torso and, with measured decelerations of 1.5-2.5G, was actively being slowed down by the neck. The torso had just about achieved the vehicle's velocity or slightly greater, regained its normal forward curve, and moved relatively downward with respect to the seat back. In addition, active control of the head's position appeared to be regained as the head was starting to approach the over the top position at about the 280-320ms point.

Phase 4-Head deceleration/Torso rest. 300-400ms: The head continued moving forward relatively faster than the shoulders, but was being decelerated by the cervical muscles. At the same time, the torso and the lower body began to achieve their post-impact rest positions during this period.

Phase 5-Restitution Phase. 400-600ms: By this time, the test subjects had just about achieved the vehicle's total impact-related velocity change and was returning to their pre-impact positions.

McConnell et al[3] stated that the results of this study reinforced and supported the findings of their previous study[97]. Their results revealed a significant range of physiological cervical extension; and therefore, no cervical hyperextension was observed. As a result, they indicated that the whiplash type symptoms that some of the participants experienced were not due to hyperextension.

Ono et al[4] conducted simulated rear-end impacts at a maximum velocity of 9km/hr. They described the kinematics of head and neck in similar phases to those of McConnell et al[3]. Szabo et al[2] described the human kinematics in impacts at 16 km/hr. They also observed a rearward motion followed by a forward motion. The rearward motion was arrested by contact between the upper torso and seat back/head restraint, while the forward motion, was arrested by the restraint system. The rearward motion occurred within 110 and 170ms while the forward motion ranged from 150 and 230ms. The kinematics for all subjects in the rearward motion was very similar, whereas in the forward motion, occupant responses began to diverge somewhat. Ono et al[4]'s findings revealed no motion of the mandible relative to the head. This finding is in agreement with that of West et al[41], who noted the absence of

mandible motion relative to the head. Their results also revealed that the translational and rotational motion of all the occupants occurred exclusively in the sagittal plane, and no significant lateral translational motion was observed. They also reported that a large component of the forward motion of the occupants within the vehicle was due to the elastic behaviour of the seat back rather than to the deceleration of the vehicle.

Some of the findings reported by Siegmund et al[25] were in agreement with those of previous studies. They reported the rearward- forward motion of the occupants, and none of the subjects exceeded their range of cervical extension motion. However, the timing values for the kinematics events were earlier than in previous studies. They also reported contact of the occupant heads with the head restraints in all participants except one. Differing from the observations of Szabo et al[2], the head of some subjects never rotated rearward of its initial "anatomically flexed" position relative to the C7-T1 joint axis. Siegmund et al[25] suggested that differences in the peak head acceleration values might be due to differences between pre-impact muscle contraction, collision severity, seat backs, and head restraints. They also reported kinematic gender differences; however, in a later study[98], they suggested that this early conclusion was due to a gender-based difference in height rather than gender per se.

### 1.2.6 Osteoligamentous cervical model

Panjabi et al[99] designed a cervical osteoligamentous model in order to study the effects of whiplash trauma on the cervical spine. This model used a head surrogate instead of a natural head, and it lacked cervical muscles. Using this model,

Panjabi et al[54] identified the injury threshold and sites of whiplash injury to the cervical spine. They used the incremental trauma method to produce injuries of increasing severity in eight human cadavers. They reported that 4.5g is the injury threshold acceleration level. The initial injuries produced at this level of acceleration were detected only by a flexibility test, and they could not be visualized at that time. Their findings suggested that the lower level of the spine, specifically C5-C6, is likely the initial injury site. At higher levels of acceleration, the upper level of the cervical spine is also prone to injury. They suggested that these injuries at the lower level of the cervical spine would most likely tear the anterior structures, such as the anterior longitudinal ligament and disc, and would damage the posterior element, specifically the facet joint.

Panjabi et al[27], using the same model, quantified the intervertebrael rotations during experimental whiplash trauma and the functional injuries to each level of the cervical spine. They also measured the dynamic elongations of the vertebrael artery and capsular ligaments, and imaged the actual injuries that occurred. Based on their findings, he proposed a bi-phasic kinematic response of the cervical spine to whiplash trauma.

The first kinematic phase occurs within the first 50-75ms and the main event of this phase is the formation of the S-shaped curvature of the cervical spine with the upper levels flexed and the lower ones extended. In the second phase, during the 100-125ms time period, all levels of the cervical spine were extended and the head reached its maximum extension at about 100ms. These findings were in agreement with those of Grauer et al[9].

Functional radiographs showed the closeness of the C5 and C6 spinous processes and disruption of the C5-C6 disc; these images indicated anterior injury at this level and suggested that injury of the facet joint might also have occurred. Flexibility testing revealed significant increases in the extension range of motion and neutral zone at the C5-C6 level after 4.5g and higher traumas. Vertebral artery elongation was significantly correlated with the horizontal acceleration of the sled, and the maximum dynamic elongation occurred during the initial S-shaped phase of whiplash.

### 1.2.7 Correlation between in vivo and human cadaver studies

The majority of the human cadaver studies[9 26 54 99 100] and studies using human volunteers[4 11 101 102] have described the formation of an S-shape of the cervical spine in simulated rear-end impacts at low velocity (Figure 1.2). They have proposed that the lower cervical spine is an initial site of injury in whiplash trauma. However, none of these studies have described what actual movement occurs in the cervical vertebras.

Kaneoka et al[11] studied, in vivo, the cervical vertebrael motion during simulated whiplash load. They observed three different patterns of movements: extension only (one subject), flexion-extension (five subjects), and no extension (four subjects). In the flexion-extension pattern, C6 rotated more quickly than the other regions and reached a plateau after approximately 100ms. Due to the early extension and forward motion of C6, the cervical spine was in a flexion position before 100ms (initial flexion); afterwards, the cervical spine undergoes an S-shaped configuration,

in which the lower cervical segments are extended and the upper segments are flexed. At approximately 150ms, while the bending moment to the neck reached its peak value, C5-C6 segment, located at the convex of this S curvature, showed the greatest extension compared with other motion segments, and this motion segment assumed an appearance similar to an open-book motion (Figure 1.3). It has been reported that the cervical spine as a whole does not exceed physiological limits of posterior rotation during the kinematics of the head-neck complex upon impact [3 25 97]; however, it has been observed that lower cervical segments exceed physiological limits of rearward rotation[9].

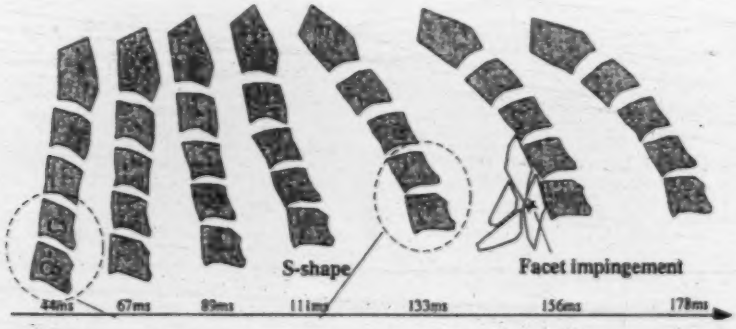


Figure 1.2: In situ Cervical Whiplash motion. Around 111ms after impact, the cervical spine shows an S-shaped curvature. Extracted from Possible zygapophyseal joint injury mechanism during whiplash loading. Kaneoka K, Inami S and Hayasaki[102]

Bogduk[103] indicated that this finding represented a hallmark in whiplash injury mechanism studies; the lower cervical segment rotates about an abnormally high IAR (Figure 1.4). This abnormally high location of the axis indicates that the vertebra undergoes no translation, its movement is purely posterior rotation. Thus, as the vertebra spin, its anterior elements separate from, while the posterior elements crush into, the vertebra below. This mechanism suggested that the resultant lesions

should be tears at the annulus and fractures at the zygapophysial joints or contusions of their meniscoids.

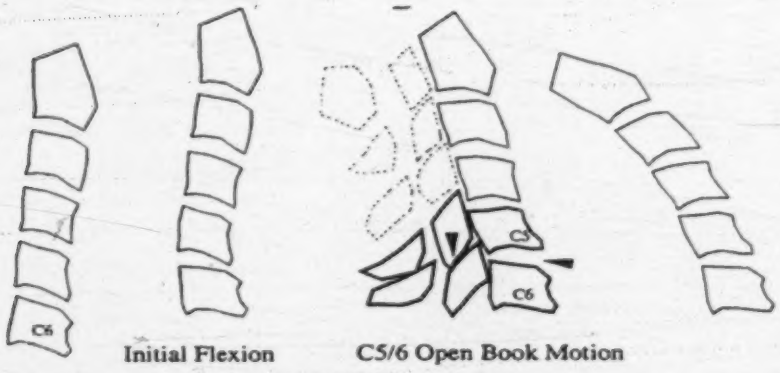


Figure 1.3: Open book motion: At approximately 150ms, the C5/6 motion segment, located at the convex of this S- curvature, showed the greatest extension compared to the other motion segments. This motion segment is demonstrated like an "open book motion." Extracted from Motion analysis of Cervical Vertebraee during bwhiplash loading, Kaneoka K, Ono K, Inami S, and Hayasaki K[101]

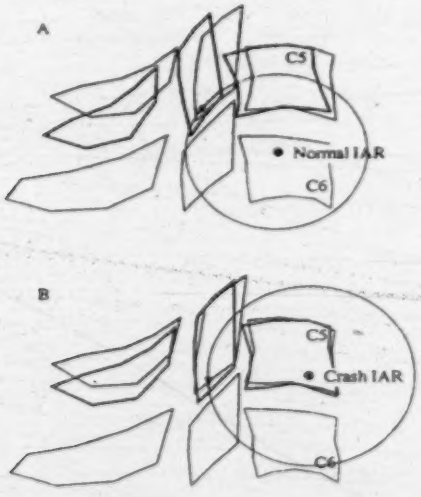


Figure 1.4: Instantaneous axis of rotation A In the normal extension motion, the C5 inferior articular facet surface rotates and slides smoothly around Normal Instantaneous Axis of Rotation. B: In the impact motion, the posterior edge of the C5 inferior articular facet shows downward movement toward the C6 facet surface and appears likely to collide with it. Extracted from Possible zygapophyseal joint injury mechanism during whiplash loading, Kaneoka k, Ono K, Inami S[102].

### 1.2.8 Electromyographic studies in simulated low velocity rear-end collisions

Magnusson et al[8] studied the cervical muscle EMG activity in low velocity rear-end impacts under expected and unexpected conditions. The cervical muscles included in this study were the SCM, trapezius, splenius capitis, semispinalis capitis, and levator scapulae. They reported that the average muscle response time for both expected and unexpected impacts from the onset of sled acceleration to the onset of EMG activity of all muscles was 112.4ms. The sequence and time lag (ms) of regions beginning to move and muscle activity in an expected case was as follows: the trunk began to move at 17ms, the head at 36ms, and the muscles at 66ms. The first muscle response occurred in the levator scapulae, then the SCM, and the trapezius muscles at average response times of 73.2ms, 73.3ms and 83.0ms respectively. The duration from onset of muscle activity to peak activity was 128.3ms. The longest duration was for the trapezius at 183.6ms and the shortest was in the SCM muscle at 42.8ms. They indicated that their results support the hypothesis of a centrally-mediated muscle response.

Castro et al[7] conducted a study to find out whether whiplash injury is caused with a $\Delta V$ between 10 and 15Km/hr. They recorded cervical muscle EMG activity; however, they did not specify which muscles were chosen nor did they present the data. They indicated that muscle response began, on average, about 60ms after the passenger compartment began to accelerate and approximately 20ms after the chest movement began. They also stated that, in all participants, the cervical muscle EMG signals started before the head movement took place.

Kumar et al[6] studied the cervical muscle EMG activity in low velocity impacts at different acceleration intensities under expected and unexpected conditions. Their results revealed that as the level of applied acceleration increased, the magnitude of the SCM EMG activity increased progressively and disproportionately; the EMG activity was also greater during the unexpected condition. They also observed that SCM exerted over 100% of the mean normalized maximum voluntary contraction, whereas the splenius and trapezius muscles never exceeded 35% of the values. They observed that the time of the sled, torso, and head acceleration onset decreased with applied acceleration. Similarly, the time of the EMG onset decreased with increased applied acceleration. Their results did not support central mediated muscle response; however, at the same time, they did not rule out the potential role of any central input.

Ono et al[4], in a similar type of study, reported that EMG activity of the paravertebral neck extensor and trapezius muscles were significantly low. The onset of the SCM muscle EMG activity ranged between 76 and 93ms with an average of 79ms. The cervical muscle activity started approximately, but no sooner than, 100-180ms before the head extension angle reached its maximum value.

### 1.2.9 Effect of anticipation in simulated low velocity rear-end collisions

Sturzenegger et al[104] performed clinical interviews, cervical spine imaging, as well as physical and neurological assessments in one hundred and thirty seven subjects who were involved in MVA and experienced whiplash injuries. Their results revealed that subjects who were aware of the imminent impact presented a lower

frequency of multiple symptoms, headaches of lower intensity, and longer latency of headache onset.

Kumar et al[5] studied the effects of anticipation in the kinematics of head and neck in simulated rear-end impacts. For males, anticipation reduced backward head acceleration by 18%, 9%, 27% and 37% for the corresponding 0.5g, 0.9g, 1.1g and 1.4g impact magnitudes. For females, anticipation reduced backward head acceleration by 9%, 20%, 34% and 29% for the corresponding 0.5g, 0.9g, 1.1g and 1.4g impact magnitudes. There was a significant two-way interaction between magnitude of impact and expectation. The reduction in head acceleration associated with anticipation of impact was more pronounced at higher impact magnitudes. This finding is in agreement with that of Ono et al[4] who reported that, when subjects intentionally tensed their muscles before 6km/hr impacts, the maximum angle of the head extension was decreased by about 30-40%. Ono et al[4] also indicated that the head and neck began to rotate sooner in those participants who activated their muscles before impact. However, Magnusson et al[8] using a 0.5g impact magnitude, did not report significant differences in reaction times between expected and unexpected impacts. Likewise, awareness of the impact did not affect the magnitude of the duration of the onset muscle activity to peak activity. The fact that Magnusson did not identify significant difference associated with awareness may be a factor of the low impact magnitude. Kumar et al[5] reported greater differences associated with expectation for larger impact magnitudes.

Recently, Siegmund et al[105] estimated the cervical muscle forces during whiplash loading based on a number of simplifying assumptions. The contraction of

the cervical muscles can generate shear, compression and bending moment in the cervical spine. The estimated shear and compression loads were 95N and 126N respectively. They reported that these shear and compression load values were within the range of computed neck loads from human subjects exposed to whiplash dynamics. Thus, they suggested that the neck muscle forces generated during a whiplash event were of sufficient magnitude to be considered in the study of whiplash biomechanics.

### 1.2.10 Electromyographic activity in patients who have experienced whiplash injuries

Nederhand et al[106] compared the cervical muscle EMG activity of a group of patients who had neck pain, stiffness or only tenderness, but no physical signs of injury (WAD grade 2, Quebec Task Force, 1995) with a group of patients who presented non-specific neck pain and with a healthy control group. Trapezius muscle EMG activity upon exercise was measured and compared in the three groups. Their results revealed only a slight increase of muscle reactivity in patients with WAD 2. There was also no consistent pattern of muscle activity level during the post exercise phase. Patients with chronic non-specific neck pain showed slightly higher pre-exercise and post-exercise muscle activity levels than those with WAD 2. Patients with WAD 2 presented greater muscle reactivity than the healthy control group. Trapezius muscle EMG activity was higher and longer in reactivity to a physical load suggesting that this muscle was involved in a vicious cycle that contributed to the persistence of pain and it was maintained by secondary myalgia.

### 1.2.11 Biomechanics of headache and neck pain in whiplash injuries

Yoganadan and Pintar[107] studied the mechanics of headaches and neck pain in whiplash using an intact human cadaver head-neck complex which included the ligaments, column, musculature, and skin. The advantages of this model are that it includes the actual masses of the head and neck as well as the occiput-atlanto joint, both of which provide information about the kinematics at this level. In addition, the inclusion of the actual head provides a more accurate location of its center of gravity. Their results revealed a reverse curvature or S-shape to the cervical head neck complex, and a single curvature of the neck at the end of the movement. They also observed a temporary occiput-C2 flexion during the acceleration phase. In contrast, the lower cervical spine facet joint demonstrated varying degrees of local compression and sliding. While the anterior and posterior regions of the facet joint slide, the posterior-most region of the joint compresses more than in the anterior-most region. These varying kinematics at the two ends of the facet joint result in a "pinching" mechanism (Figure 1.5). They suggested that this mechanism, was responsible for the neck pain reported by subjects who experience whiplash. They also proposed that the transient temporal occiput-C2 flexion was responsible for headaches.

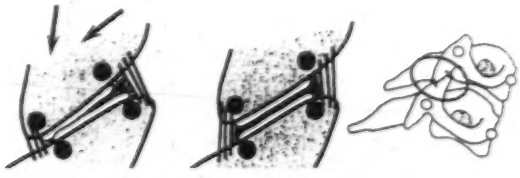

Figure 1.5: Facet joint pinching mechanism. The illustration on the left shows the lateral view of the facet joint target; middle shows the schematic of the joint prior to the impact and right shows compression (down arrow) and sliding (inclined arrow) of the joint. Extracted from Mechanics of headaches in Frontiers in whiplash trauma[107].

Cusick et al[108] indicated that scientific results from studies using this type of model should be interpreted with caution because it does not consider the active role of the cervical musculature. They suggested that inflammation or tearing of the cervical synovial intraarticular meniscoids could be part of the acute and chronic tissue changes in the facet joint resulting in nociceptive pain. The lower cervical segments could be exposed to compression forces acting on the facet joints. These forces may exceed physiologic limits, which may result in tissue damage. This damage may indicate excitation of nociceptors, lower threshold responses, and an increase in firing rates.

### 1.2.12 Zigapophyseal joints as a source of neck pain in whiplash injuries

Exhaustive research has been conducted to determine the role of the cervical zygapophyseal joints in the generation of neck pain and headaches. In the eighties, Yu et al[109] reported that the zygapophyseal joints are synovial joints with a synovial fold (meniscus). In a later study, fat pads, fibro adipose meniscus, and capsular rims were described as intraarticular inclusions of the cervical zygapophyseal joints[110]. Fibro adipose meniscus was observed in 80% of the cases. They probably serve to protect the exposed cartilage during subluxation of the joint by maintaining a film of synovial fluid between themselves and the cartilage. McLain[111] reported that the cervical facet joint contains mechanoreceptors and also fine unmyelinated nerves. The latter ones were identified in the dense capsular tissue, as well as in the synovial and aerolar tissue. He indicated that these might represent nociceptive ending fibres.

The cervical synovial joints are innervated by the dorsal cervical rami with exception of the upper cervical synovial joints. The atlantooccipital and the lateral atalantoaxial joints are innervated by the C1 and C2 ventral rami. Based on this finding, Bogduk[112] proposed that these nerves were targets that blocked the sensations coming from the zygapophysial joints.

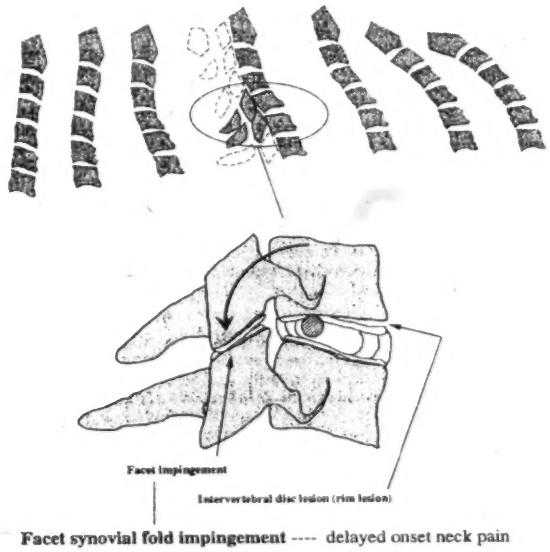


Figure 1.6: Impingement of the synovial folds of the zygapophyseal joints. Extracted from Possible zygapophyseal joint injury mechanism during whiplash loading. Kaneoka K, Ono K, Inami S and Hayasaki K[102]

In a later study, Bogduk and Marsland[113] suggested that the cervical zygapophysial joints were responsible for idiopathic neck pain. Barnsley et al[114] indicated that cervical zygapophysial joints were the most common source of chronic neck pain after whiplash injury. Interestingly, these joints responded to local anesthetic blocks such as bupivacaine but not steroids[115]. Kaneoka et al[101] suggested that an impingement and inflammation of the synovial folds in the zygapophyseal

joints were responsible for the neck pain (Figure 1.6). This mechanism is very similar to the mechanism of neck pain proposed by Yogananadan and Pintar[107].

Aprill and Bogduk[116] highlighted the importance of considering the zygapophysial joints as a potent source of neck pain. They reported that between 26% and 63% of patients might have presented zygapophyseal joint pain as the source of their intractable neck pain. Lord et al[117] conducted a double-blind clinical evaluation of the effectiveness of local anesthetic for blocking pain arising from the zygapophyseal joints. The sixty-eight participants were patients who experienced chronic neck pain and/or headaches as a consequence of whiplash trauma. Their results revealed that the more common locations for symptomatic joints were C2-C3 and C5-C6. Their results revealed a 60% prevalence of cervical zygapophysial joint pain. Recently, Slipman et al[118] also observed that articular zygapophysial joint injections were effective in the treatment of headaches originating in the C2-C3 joint after a whiplash trauma.

### 1.2.13 Role of the cervical facet joint capsule in neck pain

Recent research findings have suggested that the human cervical facet capsule may play a role in the mechanism accounting for whiplash injury[119-122]. The cervical facet capsule has neural receptors in the facet capsular ligament[111]. In addition, the capsular ligament might be loaded by spinal motion and contraction of the surrounding cervical musculature.

Winkelstein et al[120] reported that approximately 22.4% of the cervical capsule area was covered by muscle insertion. They reported that the estimation of the load to

the cervical facet capsule due to eccentric muscle contraction might be as high as 51N. If these forces were added to those acting on the capsule ligament, these forces might be as high as 66N. Interestingly, their findings revealed a significantly greater mean percentage muscle in male donors than in females; this finding supported the possibility of muscle mediated facet capsule ligament injury. However, this finding did not provide an anatomical basis for the clinical and epidemiological data which report that the C5 –C6 level is the more common site of mechanism pain than the C4-C5, nor did it find a basis for the increased frequency of whiplash related neck pain in women over men.

Siegmund et al[121] reported that the cervical facet capsular ligaments might be injured under whiplash-like loads of combined shear, bending, and compression. Winkelstein et al[119] suggested that it is unlikely that patients, who are involved in low velocity rear-end impacts, experience gross failure of the capsular ligament. However, they indicated that some vehicle occupants might be at risk of developing non-catastrophic capsule injury and perhaps the development of pain in the presence of a cervical pre-twist. Winkelstein and Myers[122] indicated that cervical muscle forces could not, by themselves, cause cervical facet capsular sub-catastrophic injury, but these in conjunction with joint motion might develop pain in the cervical facet joints.

### 1.2.14 Factors that may influence the biomechanics of whiplash injuries

Ono et al[123] indicated that it is difficult to assess the occurrence of minor neck injuries as a result of MVA using impact velocity as the only parameter. Their findings revealed that the occupant's sitting posture, cervical muscular forces, and the

presence and height of the headrests might influence neck response. Siegmund et al[124] reported that the production of symptoms in whiplash injury might be influenced not only by the kinematic response, but also by other factors such as injury tolerance of biological tissue and pain perception.

Sturzenegger et al[104] reported that the manifestation, severity, and multiplicity of initial symptoms and some of the signs of whiplash were influenced by the position of the patient's head at the moment of the impact, the type of collision, and the state of preparedness for the collision. They reported that rotated and inclined head positions led to a significantly higher frequency of multiple of symptoms, increased neck pain and headache intensity, and showed a trend to shorter latency of headache onset. In a later study, Sturzenegger et al[56] reported that head positions were significantly associated with persistence of whiplash symptoms.

Burgess et al[51] reported that TMJ or masticatory muscle injury, as a result of MVA, may be associated with varying crash characteristics including velocity, and direction of the impact, and the amount of vehicular damage. In a later study, Kolbinson et al[67] indicated that patients who were involved in rear-end collisions received more treatments and reported significantly more masticatory myalgia at the final examination compared with those subjects involved in front-end collisions. Sturzenegger et al[104] stated rear-end collisions led to a higher frequency of multiple symptoms, cranial nerve or brainstem symptoms, and unsteadiness, and significantly shorter latency of headache onset. Pennie and Agambar[125] reported that patients who were involved in stationary/rear-end collisions were more likely to have predominantly anterior tenderness; however, they did not provide more specifics

about localization of the tenderness. They also did not report significant differences between the direction of the impact and the time to recover from those symptoms or type of signs and symptoms.

Headrests were designed to prevent the neck hyperextension by blocking head motion. Norris and Watt[69] reported no significant differences in the severity of neck pain between patients who used a headrest at the moment of the impact and patients who did not use it. Maimaris et al[63] indicated that the use of a head restraint had poor effect in the presentation and severity of symptoms in a patient population that experienced whiplash injuries. However, they suggested that this might be due to the fact that most of the headrests were not properly positioned at the time of the impact.

Recently, Panjabi et al[27] suggested that lower cervical spine injuries occur before the neck is hyper extended. In other words, headrests are ineffective in preventing the formation of the S-shaped curvature of the cervical spine in the first phase of trauma when the neck injuries occur. If this finding were applicable to actual whiplash trauma, it might explain, to a certain extent, the lack of success in significantly reducing whiplash injuries following the introduction of the headrest.

### 1.3Summary and conclusions

Whiplash injuries represent the classical example of soft tissue injury to the neck.[22] Whiplash injuries have had significant economical consequences in most industrialized countries[7,19,20] however, good quality scientific research is limited[17,21]. Different types of cervical models developed with human volunteers, human

cadavers, and mathematical equations, have been used to gain a better understanding of whiplash injury.

Investigations using osteoligamentous models have reported the threshold and the initial site of injury at the cervical spine.[99] It has been determined that 4.5g is the injury threshold acceleration level,[54] and the lower levels of the spine, specifically C5-C6, is likely the initial site of injury.

Studies using human volunteers have described the kinematics of neck and head upon simulated rear-end impacts.[3, 25, 97] It has been reported that upon impact, the pelvis pulled the torso forward, then the thoracic and the cervical spine became straightened out.[3] Approximately, sixty miliseconds later, the cervical spine assumes an S-shaped configuration, in which the lower cervical segments are extended and the upper segments are flexed.[4, 11, 101, 102] This S-shaped fromation is in agreement with in vitro studies.[9, 54, 99, 100] It has also been reported that the head presents a rearward movement followed by a forward movement,[3,4,7, 8, 25, 97, 124, 126, 127], and hyperextension of the neck has not been reported.[3, 7, 25, 97]

It has been reported that it would be unlikely that significant injury results at a $\Delta V$ of 9 km/hr or less.[2, 3, 7, 41] Neck pain is the most common complaint of patients who have experienced whiplash injuries;[58-60, 64, 67] headaches represent the second most frequent symptom in subjects exposed to whiplash trauma,[68, 71, 72] followed by TMJ sign and symptoms.[67, 76, 77]

It has been suggested that damage to the soft tissues of the cervical spine might be responsible for the neck pain; structures such as the facet joint,[113, 116, 128] capsule, and ligaments[10, 119-122] are a few examples. A "pinching" mechanism for

headaches has been proponed,[20, 107] and the causation of temporomandibular joint injury is still controversial.[75, 77, 82]

Recently, the role of the cervical muscles in whiplash injury has been extensively investigated.[6, 8, 24, 25] An increase of the cervical muscle EMG activity with an increase of the impact has been reported.[6, 126] Findings regarding the onset of muscle activity, influence of gender in muscle response, how awareness of an impending impact might influence cervical muscle EMG activity, and the temporal relationship between muscle response and acceleration of the head have been reported[6, 8, 126] but warrant further investigation.

The following conclusions can be drawn from this literature review:

- Whiplash injuries represent the typical example of soft tissue injury to the neck.
- Neck pain and headaches represent the classical symptoms reported by subjects who have experienced whiplash injuries
- Despite the several methodologies used in the study of the biomechanics of whiplash event, the following agreements have been reported:
    - Upon impact, the torso moved forward; whereas, due to inertia, the head moved backward.
    - In vivo and in vitro studies have described an S-shaped configuration of the cervical spine during the whiplash event
    - Damage to the soft tissue of the cervical spine might be responsible for the neck pain reported by subjects exposed to whiplash trauma.
    - Cervical muscles likely play a role in whiplash injuries.

## 1.4 Statement of the Problem

The biomechanical mechanism accounting for whiplash injury is still controversial. Kumar et al[6] reported that responses from cervical muscles were greater with higher levels of acceleration. Likewise, Brault et al[126] indicated that their findings of higher EMG muscle activity with an increased vehicle velocity change suggested that the severity of muscle damage which might occur in a rear-end impact might increase with impact severity.

The cervical muscles control the spatial position of the head, and these muscles are the first in the line of defence for the cervical region.[6] Ono and Kano[123] reported that the occupant's tension (neck muscle tone) was one of the factors that influences the severity of neck injuries in low impact speed rear-end collisions. Castro et al[7] reported that in ΔV 8.3-10.6 km/hr rear-end impacts, EMG signals of the neck muscles started before the head movement took place. Magnusson et al[8] reported activation of the levator scapulae 73.2ms after impact (0.5g). Brault et al[126] reported that the onset of the cervical muscle EMG activity was 83ms at a ΔV 8 km/hr. Ono et al[4] reported an onset of muscle activity at 70ms (ΔV 6 km/hr) while Szabo et al[24] reported it to be approximately 122ms at a ΔV 16 km/hr. Brault et al[126] stated that differences in the reports of the cervical muscle onset EMG activity could be accounted for by the fact that some of the onset times reported in these studies were relative to the start of passenger compartment acceleration while others were relative to the bumper contact.

Gender and role of awareness in head kinematics and SCM EMG responses have been reported by several authors without a clear consensus. Brault et al [126] in

sample of 42 subjects (21 male) and ΔV 4km/hr and 8km/hr unanticipated impacts, reported gender difference in timing of onset of EMG but no gender difference for magnitude of EMG response. Magnusson et al[8] tested a sample of 8 male subjects with expected and unexpected 0.5g impacts. There was no difference in timing of onset of head movement and no difference in EMG onset or peak EMG magnitude. Kumar et al [5] in a sample of 5 male and 9 female subjects, reported gender and expectation influenced peak backward head acceleration. Kumar et al[6] reported no gender difference in EMG response in their study involving 7 subjects. For further analysis of expectancy male and female data was combined. They reported reduced peak EMG magnitude and reduced latency times with expectancy. Seigmund et al[127] reported on a sample size of 31 male and 35 female subjects exposed to 1.5g impacts. They differentiated expectancy into alerted (countdown to impact), unalerted (told impact would occur within 60 seconds) and surprised (deceived). Onset of head acceleration was not related to gender or state of expectancy. Activation of SCM occurred earlier in alerted and unalerted than in surprised events. The amplitude of SCM EMG in the surprised state had a strong gender difference, with males having higher peak EMG response. Peak head acceleration, backward head displacement and angular acceleration showed awareness-gender interaction. Females showed larger backward head movement in the surprised events.

Seigmund et al[127] suggested that the alerted and unalerted conditions reported in his study were similar to the expected and unexpected conditions reported by Kumar et al.[5,6] In the Kumar study all subjects were aware that there would be an impact with the expected group being specifically warned when the impact would

occur. Seigmund et al[127] suggest that event awareness facilitates the sensorimotor system and is more important than temporal awareness. Seigmund et al[127] also suggest the use of a molded plastic chair rather than an automobile seat may not allow direct comparison with the Kumar et al data.[5,6]

The purpose of the investigation was to analyze the SCM muscle response and head kinematics upon simulated low velocity rear-end impacts. The SCM muscle response and kinematic of head movement will be analyzed upon two levels of impact magnitudes. Gender will be entered into the analysis to determine its role in muscle response and in head acceleration. Onset and peak times of the electromyogram and acceleration peaks will be compared to determine the temporal relationship between muscle response and kinematics of head movement. Electromyogram, head acceleration and head angular displacement peaks will be compared between the unexpected (unalerted) and expected (alerted) condition to determine how awareness influences the muscle response and/or kinematics of head movement.

## 1.5 Statement of Objectives

Is there an increase in the peak head acceleration and in the sternocleidomastoid muscle EMG activity with increased accelerations upon simulated rear-end impacts?

Is there a gender difference in SCM muscle response or kinematics of the head upon simulated rear-end impacts?

Does awareness of a simulated impact influence SCM muscle response and kinematic events of the head?

Is there any temporal relationship between SCM muscle response and kinematics of the head upon simulated rear-end impacts?

## 1.6 Research hypotheses

$H_0$: There is no increase in the peak head acceleration and in the sternocleidomastoid muscle EMG activity with increased accelerations upon simulated rear-end impacts.

$H_a$: There is an increase in the peak head acceleration and in the sternocleidomastoid muscle EMG activity with increased accelerations upon simulated rear-end impacts.

$H_0$: There is no gender difference in SCM muscle response or kinematics of the head upon simulated rear-end impacts.

$H_a$: There is gender difference in SCM muscle response or kinematics of the head upon simulated rear-end impacts.

$H_0$: Awareness of a simulated impact does not influence SCM muscle response and kinematic events of the head.

$H_a$: Awareness of a simulated impact does influence SCM muscle response and kinematic events of the head.

$H_0$: There is no temporal relationship between SCM muscle response and kinematics of the head upon simulated rear-end impacts.

$H_a$: There is a temporal relationship between SCM muscle response and kinematics of the head upon simulated rear-end impacts.

## 1.7 References


1. Otremski I, Marsh JL, Wilde BR, McLardy Smith PD, Newman RJ. Soft tissue cervical spinal injuries in motor vehicle accidents. Injury 1989; 20(6):349-51.

2. Szabo T, Welcher J, Anderson R *et al*. Paper 940532 Human occupant kinematic response to low speed rear-end impacts. Thirty Eighth Stapp Car Crash Conference. Warrendale, Pennsylvania, USA: Society of Automotive Engineers., 1994.

3. McConnell W, Howard RP, Poppel JV *et al*. Paper 952724 Human head and neck kinematics after low velocity rear-end impactsUnderstanding "whiplash". 39th Stapp Car Crash Conference . Warrendale, Pennsylvania: Society Automotive Engineers, Inc, 1995: 3106-29.

4. Ono K, Kaneoka K, Wittek A, Kazjer J. Paper 973340 Cervical Injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Annual Stapp Car Crash Conference . Warrendale , Pennsylvania, USA: Society of Automotive Engineers, Inc., 1997: 339-56.

5. Kumar S, Narayan Y, Amell T. Role of awareness in head-neck acceleration in low velocity rear-end impacts. Accid Anal Prev 2000; 32(2):233-41.

6. Kumar S, Narayan Y, Amell T. An electromyographic study of low-velocity rear-end impacts. Spine 2002; 27(10):1044-55.

7. Castro WH, Schilgen M, Meyer S, Weber M, Peuker C, Wortler K. Do "whiplash injuries" occur in low-speed rear impacts? Eur Spine J 1997; 6(6):366-75.

8. Magnusson ML, Pope MH, Hasselquist L *et al*. Cervical electromyographic activity during low-speed rear impact. Eur Spine J 1999; 8(2):118-25.

9. Grauer J, Panjabi MM, Cholewicki J, Nibu K, Dvorak J. Whiplash produces an S-shaped curvature of the neck with hyperextension at lower levels. Spine 1997; 22(21):2489-92.

10. Panjabi MM, Cholewicki J, Nibu K, Grauer J, Vahldiek M. Capsular ligament stretches during in vitro whiplash simulations. J Spinal Disord 1998; 11(3):227-32.

11. Kaneoka K, Ono K, Inami S, Hayashi K. Motion analysis of cervical vertebrae during whiplash loading. Spine 1999; 24(8):763-9; discussion 770.

12. Kolbinson DA, Epstein JB, Burgess JA, Senthilselvan A. Temporomandibular disorders, headaches, and neck pain after motor vehicle accidents: a pilot investigation of persistence and litigation effects. J Prosthet Dent 1997; 77(1):46-53.

13. Kolbinson DA, Epstein JB, Burgess JA. Temporomandibular disorders, headaches, and neck pain following motor vehicle accidents and the effect of litigation: review of the literature. J Orofac Pain 1996; 10(2):101-25.

14. Burgess JA, Dworkin SF. Litigation and post-traumatic TMD: how patients report treatment outcome. J Am Dent Assoc 1993; 124(6):105-10.

15. Crowe DE. A new diagnostic sign in neck injuries. California Medicine 1964; 100(1):12-3.

16. Ferrari R. The medical history of whiplash. In : Ferrari R, editor. The whiplash encyclopedia: The facts and myths of whiplash . United States of America: Aspen Publishers Inc, 1999: 17-21.

17. Spitzer WO, Skovron ML, Salmi LR *et al*. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its

management. Spine 1995; 20(8 Suppl):1S-73S.

18. Barnsley L, Lord S, Bogduk N. The pathophysiology of whiplash. In. Spine: State of Art Reviews. Vol. 12. Hanley &Belfus Inc, 1998: 209-42.

19. Malleson A. Chronic whiplash syndrome. Psychosocial epidemic. Canadian Family Physician 1994; 40:1906-9.

20. Yoganandan N, Pintar FA, Kleinberger M. Whiplash injury. Biomechanical experimentation. Spine 1999; 24(1):83-5.

21. Teasell R and Shapiro A. Whiplash injuries: an update. Pain Research Management 1998; 3(2):81-90.

22. Bogduk N, Yoganandan N. Biomechanics of the cervical spine Part 3: minor injuries. Clin Biomech (Bristol, Avon) 2001; 16(4):267-75.

23. Panjabi MM. Cervical spine models for biomechanical research. Spine 1998; 23(24):2684-700.

24. Szabo T, Welcher J. Paper 962432 Human subject kinematics and electromyographic activity during low speed rear impacts. 40th Stapp Car Crash Conference. Warrendale, Pennsylvania, USA: Society of Automotive Engineers Inc. 1996: 295-314.

25. Siegmund G, King D, Lawrence J, Wheeler J, Brault J and Smith T. Paper 973341 Head/neck kinematic response of human subjects in low-speed rear-end collisions. 41st Annual Stapp Car Crash Conference. Warrendale, Pennsylvania, USA : Society Automotive Engineers, Inc, 1997: p 357-85.

26. Luan F, Yang KH, Deng B, Begeman PC, Tashman S, King AI. Qualitative analysis of neck kinematics during low-speed rear-end impact. Clin Biomech (Bristol,

Avon) 2000; 15(9):649-57.

27. Panjabi MM, Cholewicki J, Nibu K, Grauer JN, Babat LB, Dvorak J. Mechanism of whiplash injury. Clin Biomech (Bristol, Avon) 1998; 13(4-5):239-49.

28. Penning L. Acceleration injury of the cervical spine by hypertranslation of the head. Part I. Effect of normal translation of the head on cervical spine motion: a radiological study. European Spine Journal 1992; 1:7-12.

29. Panjabi M. Summary: Cervical spine models for biomechanical research. Spine 1998; 24(23):2699-770.

30. May B, Saha S, Saltzman M. A three-dimensional mathematical model of temporomandibular joint loading. Clin Biomech (Bristol, Avon) 2001; 16(6):489-95.

31. Koolstra JH, van Eijden TMGJ. Biomechanical analysis of jaw-closing movements. J. Dent REs 1995; 74(9):1564-70.

32. Koolstra JH, van Eijden TM. A method to predict muscle control in the kinematically and mechanically indeterminate human masticatory system. J Biomech 2001; 34(9):1179-88.

33. Schneider K, Zernicke RF, Clark G. Modeling of jaw-head-neck dynamics during whiplash. J Dent Res 1989; 68(9):1360-5.

34. Jonsson H Jr, Bring G, Rauschning W, Sahlstedt B. Hidden cervical spine injuries in traffic accident victims with skull fractures. J Spinal Disord 1991; 4(3):251-63.

35. Taylor JR, Twomey LT. Acute injuries to cervical joints. An autopsy study of neck sprain. Spine 1993; 18(9):1115-22.

36. Bogduk N. An Overview of Whiplash. In: Yogandan N, Pintar FA. Frontiers in

Whiplash. IOS PRESS, 2000: 3-9.

37. Kaiser J and Holland B. Imaging of the Cervical Spine. Spine 1998; 23(24):2070-712.

38. Dall'Alba PT, Sterling MM, Treleaven JM, Edwards SL, Jull GA. Cervical range of motion discriminates between asymptomatic persons and those with whiplash. Spine 2001; 26(19):2090-4.

39. Osterbauer PJ, Long K, Ribaudo TA *et al*. Three-dimensional head kinematics and cervical range of motion in the diagnosis of patients with neck trauma. J Manipulative Physiol Ther 1996; 19(4):231-7.

40. Bogduk N, Mercer S. Biomechanics of the cervical spine. I: Normal kinematics. Clin Biomech (Bristol, Avon) 2000; 15(9):633-48.

41. West Dh, Gough PE, Harper PE. Low speed rear-end collision testing using human subjects. Accident Reconstruction Journal 1993; 5(3):22-6.

42. Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical response of human subjects to rear-end automobile collisions. Arch Phys Med Rehabil 1998; 79(1):72-80.

43. Ferrari R. Letter to the Editor: Rear-end auto collisions. Arch Phys Med Rehabil 1998; 79(6):721; discussion 722-3.

44. Kwan O. Rear-end auto collisions. Arch Phys Med Rehabil 1998; 79(6):721-3.

45. Castro WH, Meyer SJ, Becke ME *et al*. No stress--no whiplash? Prevalence of "whiplash" symptoms following exposure to a placebo rear-end collision. Int J Legal Med 2001; 114(6):316-22.

46. Versteegen GJ, Kingma J, Meijler WJ, ten Duis HJ. Neck sprain after motor vehicle accidents in drivers and passengers. Eur Spine J 2000; 9(6):547-52.

47. Pearce JM. Headaches in the whiplash syndrome. Spinal Cord 2001; 39(4):228-33.

48. Weiss HD, Stern BJ, Goldberg J. Post-traumatic migraine: chronic migraine precipitated by minor head or neck trauma. Headache 1991; 31(7):451-6.

49. Braun B, DiGiovanna A, Schiffman E, Bonnema J. A cross-sectional study of temporomadnibular joint dysfucntion in post-cervical trauma patients. Journal of Craniomandibular Disorders: Facial and Oral Pain 1992; 6:24-31.

50. Burgess J. Symptom characteristics in TMD patients reporting blunt trauma and/or whiplash injury. J Craniomandib Disord 1991; 5(4):251-7.

51. Burgess JA, Kolbinson DA, Lee PT, Epstein JB. Motor vehicle accidents and TMDs: assessing the relationship. J Am Dent Assoc 1996; 127(12):1767-72.

52. Radanov B and Dvorak J. Impaired Cognitivge functioning after whiplash injury of the cervical spine. Spine 21(3):392-7.

53. Gargan M, Bannister G, Main C, Hollis S. The behavioural response to whiplash injury. J Bone Joint Surg Br 1997; 79(4):523-6.

54. Panjabi MM, Nibu K, Cholewicki J. Whiplash injuries and the potential for mechanical instability. Eur Spine J 1998; 7(6):484-92.

55. Ritcher M, Otte D, Pohlemann T, Krettek C, Blauth M . Whiplash-type neck distortion in restrained car drivers: frequency, causes and long term results. European Spine Journal 2000; 9:109-17.

56. Sturzenegger M, Radanov BP, Di Stefano G. The effect of accident mechanisms and initial findings on the long-term course of whiplash injury. J Neurol 1995; 242(7):443-9.

57. Suissa S, Harder S, Veilleux M. The relation between initial symptoms and signs and

the prognosis of whiplash. Eur Spine J 2001; 10(1):44-9.

58. Barnsley L, Lord S, Bogduk N. Whiplash injury. Pain 1994; 58(3):283-307.

59. Hammacher ER, van der Werken C. Acute neck sprain: "whiplash' reappraised. Injury 1996; 27(7):463-6.

60. Squires B, Gargan MF, Bannister GC. Soft-tissue injuries of the cervical spine. 15-year follow-up. J Bone Joint Surg Br 1996; 78(6):955-7.

61. Borchgrevink GE, Lereim I, Royneland L, Bjorndal A, Haraldseth O. National health insurance consumption and chronic symptoms following mild neck sprain injuries in car collisions. Scand J Soc Med 1996; 24(4):264-71.

62. Hohl M. Soft-tissue injuries of the neck in automobile accidents. Factors influencing prognosis. J Bone Joint Surg Am 1974; 56(8):1675-82.

63. Maimaris C, Barnes MR, Allen MJ. 'Whiplash injuries' of the neck: a retrospective study. Injury 1988; 19(6):393-6.

64. Karlsborg M, Smed A, Jespersen H *et al*. A prospective study of 39 patients with whiplash injury. Acta Neurol Scand 1997; 95(2):65-72.

65. Kasch H, Stengard-Pedersen K, Arendt-Nielsen L, Jensen T. Headache, neck pain, and neck mobility after acute whiplash injury. Spine 2001; 26(11):1246-51.

66. Friedman MH, Weisberg J. The craniocervical connection: a retrospective analysis of 300 whiplash patients with cervical and temporomandibular disorders. Cranio 2000; 18(3):163-7.

67. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. Effect of impact and injury characteristics on post-motor vehicle accident temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998; 85(6):665-73.

68. Magnusson T. Extracervical symptoms after whiplash trauma. Cephalgia 14:223-7.

69. Norris SH, Watt I. The prognosis of neck injuries resulting from rear-end vehicle collisions. J Bone Joint Surg Br 1983; 65(5):608-11.

70. Steigerwald DP, Verne SV, Young D. A retrospective evaluation of the impact of temporomandibular joint arthroscopy on the symptoms of headache, neck pain, shoulder pain, dizziness, and tinnitus. Cranio 1996; 14(1):46-54.

71. Balla J, Karnaghan J. Whiplash headache. Clin Exp Neurol 1987; 23:179-82.

72. De Benedittis G, De Santis A. Chronic post-traumatic headache: clinical, psychopathological features and outcome determinants. J Neurosurg Sci 1983; 27(3):177-86.

73. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Third occipital nerve headache: a prevalence study. J Neurol Neurosurg Psychiatry 1994; 57(10):1187-90.

74. Radanov BP, Di Stefano G, Augustiny KF. Symptomatic approach to posttraumatic headache and its possible implications for treatment. Eur Spine J 2001; 10(5):403-7.

75. Pullinger AG, Seligman DA. Trauma history in diagnostic groups of temporomandibular disorders. Oral Surg Oral Med Oral Pathol 1991; 71(5):529-34.

76. Kronn E. The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. J Orofac Pain 1993; 7(2):209-13.

77. De Boever JA, Keersmaekers K. Trauma in patients with temporomandibular disorders: frequency and treatment outcome. J Oral Rehabil 1996; 23(2):91-6.

78. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: initial signs, symptoms, and diagnostic characteristics. J Orofac Pain 1997; 11(3):206-14.

79. Probert TC, Wiesenfeld D, Reade PC. Temporomandibular pain dysfunction disorder resulting from road traffic accidents--an Australian study. Int J Oral Maxillofac Surg 1994; 23(6 Pt 1):338-41.

80. Kasch H, Hjorth T, Svensson P, Nyhuus L, Jensen TS. Temporomandibular disorders after whiplash injury: a controlled, prospective study. J Orofac Pain 2002; 16(2):118-28.

81. Heise AP, Laskin DM, Gervin AS. Incidence of temporomandibular joint symptoms following whiplash injury. J Oral Maxillofac Surg 1992; 50(8):825-8.

82. McKay DC, Christensen LV. Whiplash injuries of the temporomandibular joint in motor vehicle accidents: speculations and facts. J Oral Rehabil 1998; 25(10):731-46.

83. Westesson PL, Katzberg RW, Tallents RH, Sanchez-Woodworth RE, Svensson SA. CT and MR of the temporomandibular joint: comparison with autopsy specimens. AJR Am J Roentgenol 1987; 148(6):1165-71.

84. Westesson PL, Katzberg RW, Tallents RH, Sanchez-Woodworth RE, Svensson SA, Espeland MA. Temporomandibular joint: comparison of MR images with cryosectional anatomy. Radiology 1987; 164(1):59-64.

85. Tasaki MM, Westesson PL. Temporomandibular joint: diagnostic accuracy with sagittal and coronal MR imaging. Radiology 1993; 186(3):723-9.

86. Westesson PL. Reliability and validity of imaging diagnosis of temporomandibular joint disorder. Adv Dent Res 1993; 7(2):137-51.

87. Weinberg S, Lapointe H. Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 1987; 45(8):653-6.

88. Pressman BD, Shellock FG, Schames J, Schames M. MR imaging of temporomandibular joint abnormalities associated with cervical hyperextension/hyperflexion (whiplash) injuries. J Magn Reson Imaging 1992; 2(5):569-74.

89. Schellhas KP, Wilkes CH, Omlie MR, Block JC, Larsen JW, Idelkope BI. Temporomandibular joint imaging. Practical application of available technology. Arch Otolaryngol Head Neck Surg 1987; 113(7):744-8.

90. Garcia R Jr, Arrington JA. The relationship between cervical whiplash and temporomandibular joint injuries: an MRI study. Cranio 1996; 14(3):233-9.

91. Bergman H, Andersson F, Isberg A. Incidence of temporomandibular joint changes after whiplash trauma: a prospective study using MR imaging. AJR Am J Roentgenol 1998; 171(5):1237-43.

92. Bertram S, Rudisch A, Innerhofer K, Pumpel E, Grub-wieser, Emshoff R. Diagnosing TMJ internal derangement and osteoarthritis with magnetic resonance imaging. Journal of American Dental Association 2001; 132:753-61.

93. Steed PA, Wexler GB. Temporomandibular disorders--traumatic etiology vs. nontraumatic etiology: a clinical and methodological inquiry into symptomatology and treatment outcomes. Cranio 2001; 19(3):188-94.

94. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: treatment and outcomes. J Orofac Pain 1997; 11(4):337-45.

95. Cassidy JD, Carroll LJ, Cote P, Lemstra M, Berglund A, Nygren A. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. N Engl J Med 2000; 342(16):1179-86.

96. Severy Dm, Mathewson J.H and Bechtol MD. Controlled automobile rear-end collisions, an investigation of related engineering and medical phenomena. Canadian Services Medical Journal 1955; 727-59.

97. McConnell W, Howard RP, Guzman HM *et al*. Paper 930889 Analysis of human test subject kinematic responses to low velocity rear-end impacts. Presentation to . SAE International Congress and Exposition. Warrendale, Pennsylvania, USA: Society of Automotive Engineers, Inc , 1993: 21-30.

98. Siegmund GP, Heinrichs BE, Wheeler JB. The influence of head restraint and occupant factors on peak head/neck kinematics in low-speed rear-end collisions. Accid Anal Prev 1999; 31(4):393-407.

99. Panjabi MM, Cholewicki J, Nibu K, Babat LB, Dvorak J. Simulation of whiplash trauma using whole cervical spine specimens. Spine 1998; 23(1):17-24.

100. Cholewicki J, Panjabi MM, Nibu K, Babat LB, Grauer JN, Dvorak J. Head kinematics during in vitro whiplash simulation. Accid Anal Prev 1998; 30(4):469-79.

101. Kaneoka K, Ono K, Inami S, Hayashi K. Motion Analysis of Cervical Vertebrae during Whiplash Loading. Cervical Spine Research Society. 1997.

102. Kaneoka K, Ono K, Inami S, Hayashi K. Possible zygapophyseal joint injury mechanism duringt whiplash loading. 44th Annual Meeting, Orthopedic Research Society. 1998.

103. Bogduk N. Point of View. Spine 1999; 24(8):770.

104. Sturzenegger M, DiStefano G, Radanov BP, Schnidrig A. Presenting symptoms and signs after whiplash injury: the influence of accident mechanisms. Neurology 1994; 44(4):688-93.

105. Siegmund G, Brault J, Chimich D. Do cervical muscles play a role in whiplash injury? Journal of Whiplash and Related Disorders 2002; 1:23-40.

106. Nederhand Mj, Hermens J, Ijzerman M, Turk D, Zilvord G. Cervical muscle dysfucntion in chronic whiplash-associated disorder grade 2. Spine 2002; 27(10):1056-61.

107. Yoganandan N, Pintar F. Mechanics of headache and neck pain in whiplash. In: Pintar Frank, Yoganandan N. Frontiers in Whiplash Trauma. IOS Press: Ohmsha, 2000: 173-97.

108. Cusick J, Pintar F, Yoganandan N. Whiplash Syndrome: Kinematic factors influenccing pain patterns. Spine 2001; 26(11):1252-8.

109. Yu SW, Sether L, Haughton VM. Facet joint menisci of the cervical spine: correlative MR imaging and cryomicrotomy study. Radiology 1987; 164(1):79-82.

110. Mercer S, Bogduk N. Intra-articular inclusions of the cervical synovial joints. Br J Rheumatol 1993; 32(8):705-10.

111. McLain RF. Mechanoreceptor endings in human cervical facet joints. Spine 1994; 19(5):495-501.

112. Bogduk N. The clinical anatomy of the cervical dorsal rami. Spine 1982; 7(4):319-30.

113. Bogduk N, Marsland A. The cervical zygapophysial joints as a source of neck pain. Spine 1988; 13(6):610-7.

114. Barnsley L, Lord SM, Wallis BJ, Bogduk N. The prevalence of chronic cervical

zygapophysial joint pain after whiplash. Spine 1995; 20(1):20-5; discussion 26.

115. Barnsley L, Lord SM, Wallis BJ, Bogduk N. Lack of effect of intraarticular corticosteroids for chronic pain in the cervical zygapophyseal joints. N Engl J Med 1994; 330(15):1047-50.

116. Aprill C, Bogduk N. The prevalence of cervical zygapophyseal joint pain: A first approximation. Spine 1992; 17(7):744-7.

117. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo- controlled prevalence study. Spine 1996; 21(15):1737-44; discussion 1744-5.

118. Slipman CW, Lipetz JS, Plastaras CT, Jackson HB, Yang ST, Meyer AM. Therapeutic zygapophyseal joint injections for headaches emanating from the C2-3 joint. Am J Phys Med Rehabil 2001; 80(3):182-8.

119. Winkelstein BA, Nightingale RW, Richardson WJ, Myers BS. The cervical facet capsule and its role in whiplash injury: a biomechanical investigation. Spine 2000; 25(10):1238-46.

120. Winkelstein BA, McLendon RE, Barbir A, Myers BS. An anatomical investigation of the human cervical facet capsule, quantifying muscle insertion area. J Anat 2001; 198(Pt 4):455-61.

121. Siegmund GP, Myers BS, Davis MB, Bohnet HF, Winkelstein BA. Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading. Spine 2001; 26(19):2095-101.

122. Winkelstein B and Myers B. Cervical motion segment, combined loading, muscle

forces, and facet joint: A mechanical hypothesis for whiplash injury. In: Yoganandan N, Pintar F, Editors. Frontiers in whiplash trauma: clinical and biomechanical. Washington DC USA: IOS PRESS; 2000: 249-62.

123. Ono K, Kanno M. Influences of the physical parameters on the risk to neck injuries in low impact speed rear-end collisions. Accid Anal Prev 1996; 28(4):493-9.

124. Siegmund GP, Brault JR, Wheeler JB. The relationship between clinical and kinematic responses from human subject testing in rear-end automobile collisions. Accid Anal Prev 2000; 32(2):207-17.

125. Pennie B, Agambar L. Patterns of injury and recovery in whiplash. Injury 1991; 22(1):57-9.

126. Brault JR, Siegmund GP, Wheeler JB. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. Clin Biomech (Bristol, Avon) 2000; 15(6):426-35.

127. Siegmund GP, Sanderson DJ, Myers BS, Inglis JT. Awareness affects the response of human subjects exposed to a single whiplash-like perturbation. Spine 2003; 28(7):671-9.

128. Barnsley L, Lord S, Wallis B, Bogduk N. False-positive rates of cervical zygapophysial joint blocks. Clin J Pain 1993; 9(2):124-30.

# Chapter Two

## Research Paper

### Kinematics of Head Movement and the Role of the Sternocleidomastoid Muscle in Simulated Low Velocity Rear-end Impacts

## 2.1 Introduction

Rear-end impacts result in a higher frequency of whiplash injuries in comparison with other types of MVA[1]. More than a million whiplash injuries occur each year in the United States[2]. Although not catastrophic, these injuries have significant economic impact and are estimated to cost the United States $4.5 billion annually[3]. In Canada, Spitzer et al[4] reported that approximately 5000 whiplash cases annually accounted for 20% of traffic injury claims in Quebec.

The biomechanical mechanism accounting for whiplash injury is still controversial. Scientific findings have suggested that damage to the soft tissues of the cervical spine might be responsible for the neck pain; structures such as the facet joint[5-7], capsule, and ligaments[8-12] are a few examples. Recently, researchers have focused on the role of the cervical muscles in whiplash injury [13-17].

Kumar et al[13] suggested that the mechanism for whiplash injury is complex and progressive. They proposed a hierarchical model in which the first injury is to the muscle, followed by injury to the ligaments, the facet joints, and the brain. These steps are in sequence and related to increasing magnitude of the impact. The common perception that muscles activate too late in a rear-end collision to affect whiplash injury is incorrect[15]: Castro et al[18] reported that EMG signals of the neck muscles started before the head movement took place. However, no EMG data was presented and they did not specify which muscles were studied. Differences in onset times for cervical EMG muscle activity can be seen in the literature[14 17 19 20]; this discrepancy could be accounted for by the fact that some of the onset times reported in these

studies were relative to the start of passenger compartment acceleration while others were relative to the bumper contact[17].

It has been reported that muscle responses were greater at higher levels of acceleration[13 17]. This suggests that the severity of muscle damage which might occur in a rear-end impact increases with impact severity[17]. It has also been reported that among the cervical muscles, the SCM muscle might play a significant role in whiplash injury[13 14 17 19 21]

Data regarding the effect of expectancy on the kinematics of head and neck is also controversial. Sturzenegger et al[22] conducted clinical interviews, as well as, physical and neurological assessments in subjects who were involved in MVA and experienced whiplash injuries. Their results revealed that subjects who were aware of the imminent impact presented a lower frequency of multiple symptoms, had headaches of lower intensity, and showed longer latency of headache onset. Kumar et al[23] reported that awareness of an impact reduced the acceleration of the head. On the other hand, Magnusson et al[14] did not report significant differences in muscle reaction times between expected and unexpected conditions. On the other hand, gender differences have been reported by Siegmund et al[24]and by Brault et al[17], but not by Kumar et al[13].

As a result of the limitations mentioned above, this study was designed to determine the head kinematics and the SCM muscle response in human volunteers exposed to simulated rear-end impacts at low velocities. The objectives of this study were to evaluate:

- Behaviour of SCM activity at two impact magnitudes.

- Temporal relationship between SCM muscle activity and kinematic events of the head.
- Relationship of impact awareness with muscle response and head acceleration.
- Gender differences for muscle response and head kinematics.

## 2.2 Methods and Materials

The Human Research Ethics Board at the University of Alberta approved the protocol for this study. Forty-one individuals were screened and deemed eligible to participate in this study over a two-month period. Participants were recruited by poster advertisement on the University of Alberta campus. One male subject did not complete the experimental phase due to a panic attack type reaction. Demographic data are presented in Table 2.1.

Participants were between 18 and 35 years old, healthy and asymptomatic from any masticatory cervical or temporomandibular joint pain. Exclusion criteria included; any systemic medical condition, pregnancy, restricted cervical or mandibular movements, history of car accident or trauma to the back or neck within the last twelve months, more than one missing tooth by quadrant with the exception of the third molar, and the wearing of any type of occlusal appliance.

Participants attended two appointments. At the first appointment subjects read and signed the information sheet, completed a medical history form and gave informed consent. Clinical examination of the head and neck was performed on each subject in order to check cervical and mandibular mobility as well as absence of

symptoms. The second appointment was the experimental phase. Subjects were briefly reminded about the details of this study. Each subject underwent three impacts: two unexpected and one expected. EMG activity of the SCM muscles and kinematics of the head were recorded.

### 2.2.1 Experimental Set-up

*EMG system:* The EMG system (Delsys Inc, Boston, MA, USA) included two surface electrodes, amplifiers and a screen where the recording was displayed. The electrodes were positioned on the SCM muscles utilizing Kumar et al[13] protocol.

*Acceleration set up:* Accelerometers were used to measure acceleration of the sled, subject's torso and head.

*Acceleration sled set up*: The sled system consisted of a 250cm by 125 cm raised wooden platform, with two 200 cm long parallel tracks mounted 60 cm apart along the length of the platform. A car seat (unknown vehicle model) was sturdily mounted on a rectangular sliding board coupled with the tracks for friction-reduced travel upon impact Figure 2.1). One uniaxial accelerometer (25g) (Crossbow Technology, San José, CA) was located in the car seat to measure acceleration of the sled relative to the floor. The magnitude of the acceleration of each impact was achieved with an assembly of a pneumatic cylinder connected to an air supply through a pressure regulator calibrated for the delivery of known forces (for more details regarding the sled system refer to Kumar et al[23]).

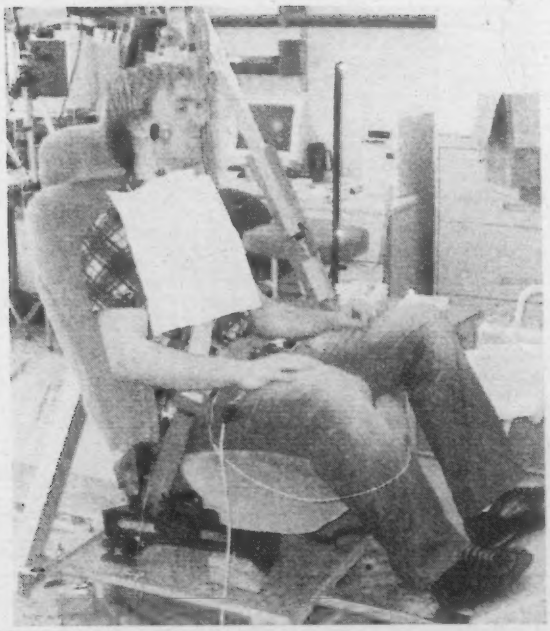

Figure 2.1: Car seat mounted in the sliding board.

*Set up of torso acceleration:* A second triaxial accelerometer (5g) (Crossbow Technology, San José, CA) was located on top of the sternum, to measure torso acceleration relative to the sled[23].

*Head acceleration set up*: A custom designed accelerometer system was developed to measure the acceleration of the head. The system included: a multipurpose circuit board SB1 (Ross Stirling, Edmonton, Alberta, Canada), a 16 channel 12 bit A/D converter, ± 5 V input range (National Instruments Corporation, Austin, TX USA), two biaxial accelerometers ± 10g (item model ADXL 210, Analog Devices, Norwood, MA USA) and 2-axes magnetoresistive sensor (item model HMC 1022, Honeywell, Morriston, NJ, USA). The accelerometer board (Figure 2.2) was attached to the maxillary teeth with a custom dental tray. A clear plastic extension

attached to the tray, positioned the accelerometer system outside of the mouth. The accelerometer was aligned with the midline of the subject.

A linear power supply Hewlet Packard (Mississagua, ON, Canada) set to 5 volts was the power source for this accelerometer board. The board was connected to the data acquisition system with ribbon cables. A headgear was used to support these cables and thus prevent their weight from interfering with the recording of head motion.

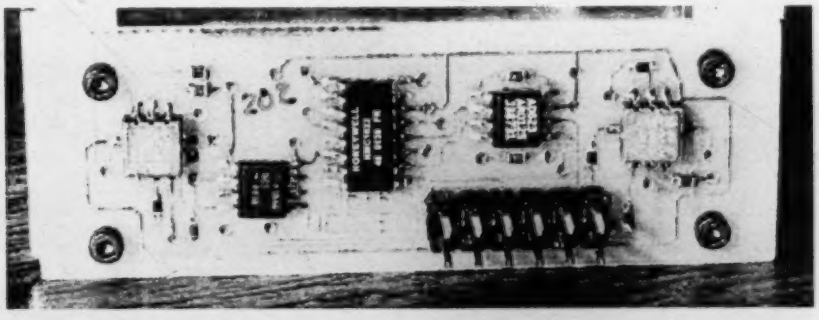

Figure 2.2: Multipurpose circuit board SB1.

*High Speed Video cameras:* Three reflectors in conjunction with three ProReflex Cameras (item model MCO 240, Qualisys, Savedalen, Sweden) with a PC reflex software (Qualisys, Savedalen, Sweden) were also used to record head motion. Two reflectors were placed on the plastic extension attached to the dental tray and one reflector was placed in the anterior temporal region of the head. The use of three markers allowed recording in 3-D. The cameras recorded at a sampling rate of 200Hz.

### 2.2.2 Data Acquisition

The directions of the global axis (Figure 2.3) were defined as follows:

- The Y-axis was defined parallel to the direction of the earth's gravity and negative down.
- The X-axis was defined as parallel to the floor with positive toward the front of the sled and the subject.
- The Z- axis refers to the medial-lateral plane.

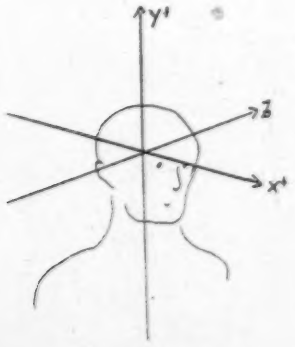


Figure2.3: Graphic representation of the global axis: Y axis was defined parallel to the direction of the earth's gravity and negative down. The X-axis was defined as parallel to the floor with positive to the front of the sled and the subject. The Z-axis refers to the medio-lateral plane.

Normalized EMG data were obtained for the right and left SCM muscles; these were expressed as a percentage of the maximum raw electromyogram peak obtained during the cervical strength test.

Angular acceleration of the head was obtained. Linear acceleration of the head in the X and Y-axes at the site of the anterior temporal region was measured. Angular displacement was determined. Angular and linear acceleration data were obtained

from the accelerometer board system; whereas, the angular displacement was obtained from the video cameras.

Onset time and peak time for the EMG and kinematic variables were determined. Onset and peak time were relative to the onset of chair movement. Onset time was defined as the time in which 5% of the magnitude value of the peak occurred. Peak time was defined as the time in which the maximum value of the variable was reached. Data acquisition was restricted to the first 750ms after impact.

### 2.2.3 Experimental Phase

The subject's skin over the SCM muscles was vigorously cleaned with a paper towel and alcohol prior to application of the electrodes. Two bipolar electrodes with an interelectrode distance of 1 cm were placed parallel to the direction of muscle fibres and over the sternal belly (C4 level) of the left and right SCM muscles.

Each participant was seated in an upright position with her/his legs uncrossed, and head straight. The participants were asked to perform a maximum flexion force using the SCM muscles. The muscle force and the EMG activity generated in this exercise were recorded for each subject[13].

The weight and height of each subject were recorded. After, weight and height measurement, subjects were prepared for impacts. The triaxial accelerometer was placed on the sternum of each participant. The custom tray with the accelerometer board and reflective balls was loaded with polyvinyl siloxane impression material (Kerr, Romulus, MI, USA) and placed in the upper jaw of each subject.

Each subject underwent three impacts. Two unexpected impacts: slow and fast, and one expected impact of the same magnitude of the unexpected fast impact. The mean chair acceleration peaks in the X, Y and Z- axes for each acceleration level are presented in Table 2.2. The peak acceleration values in the Z- axis were significantly lower than those of the X (p value 0.001, power 0.999) or Y-axis (p value 0.001, power 0.999). This confirms the assumption that the movement occurred mostly in the X and Y-axes.

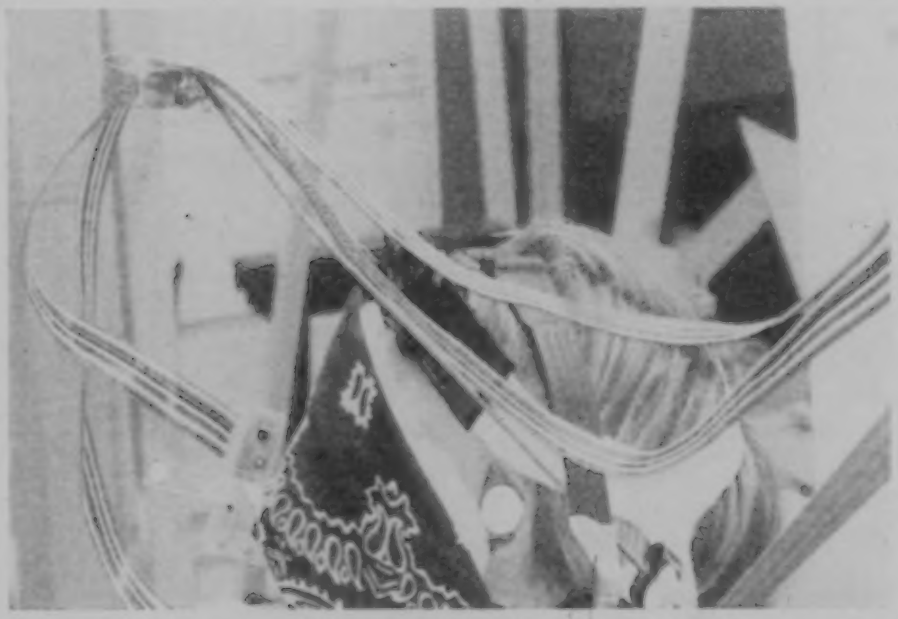

Figure 2.4: Set up of a participant for an unexpected impact. Headphones and a blindfold fabric were used to block of any clue about the imminent impact. Acceleration board and the two markers are also observed. The third marker was placed in the anterior temporal region. A headgear was used to prevent the weight of the cables from interfering with the recording of head motion.

The order of impacts was randomized. For the unexpected impacts, each subject listened to loud music and fabric blindfold was used to cover participant's eyes. Subjects were aware that there would be an impact, but were not advised of timing or impact magnitude. There was no attempt do deceive the subject with a

"surprise" impact. In the expected impact, subjects were told the magnitude of the impact in qualitative terms, ie a fast impact, and when the impact would happen.

An example of the set up for an unexpected impact is depicted in Figure 2.4. The experimental sessions lasted approximately one hour.

### 2.2.4 Data Processing

A mechanical engineer, who was not a member of this research team, processed the raw EMG, accelerometer and video camera data files.

The raw files obtained with the PCR reflex program were exported to a Tracker 3-D program (Qualisys, Savedalen, Sweden) in which the signals of the reflective balls were labelled and missing data were identified. Also, the raw files were transformed in such a way that they could be read in a text editor. These new files contained all the 3-D positional data for the three markers to a resolution of $10^{-6}$ meters. The three markers had their position monitored at a sampling rate of 200Hz for 5 seconds. The marker on the anterior temporal region was used to determine the head displacement in the X and Y-axes; whereas, the two other reflective balls, located in the extension attached to the tray, were use to determine the angular displacement. No video camera data were recorded prior to the start of the test.

The accelerometer board system incorporated a low pass filter with a frequency cut-off of 50Hz. The raw files from the accelerometer system recorded 10 seconds: five seconds pre-impact and five seconds post impact. However, a 750ms window at a sampling rate of 4000Hz with 5 seconds of pre-recorded data was

analyzed. The high frequency used for sampling these data was used for an engineering-related investigation.

The raw files from the EMG data were analyzed using the Root Mean Square technique[25]. The value from the cervical strength test was considered as the maximum value; the value from the actual impact was expressed as a percentage of the maximum value. This percentage value represents the normalized EMG data.

Files for the different impacts were coded for blinding. Missing data points and data points presenting a technical error were identified and eliminated. The original sample size of forty subjects dropped to approximately thirty for most variables, and to eighteen for a few head kinematic parameters.

Special care was taken to integrate the data files for consistent timing of impact; the two recording systems had as time 0 the firing of the pneumatic cylinder piston, which caused the acceleration of the chair. In a second step, the timing of the response variables was adjusted so the onset of acceleration of the chair represented time 0 in each impact and for each subject.

### 2.2.5 Statistical Analysis

The data was organized in an Excel spreadsheet and analyzed using SPSS (SPSS, Chicago, Il, USA). Repeated measures statistical method was used to analyze the response of the variable with an increase of the magnitude of the impact as well as its response in the expected and unexpected impacts. Gender was entered into the analysis. The same statistical method was used to determine whether the EMG muscle activity or the movement of the head was initiated in the first place as well as

to compare the timing among the kinematic events of the head. The significance level alpha .05 was used to determine the level of significance of the data.

## 2.3 Results

The video camera and accelerometer data presented a good agreement (Figure 2.5). Subjects presented a rearward movement followed by a rebound phase. The head moved backward and upward on impact. In the rebound phase, the head moved forward and downward (Figure 2.6a and Figure 2.6b).

Upon impact, the head and torso are motionless within the first 22m, then the head begins to accelerate linearly backward reaching the peak shortly after 30ms. The torso begins to accelerate linearly forward by 85ms, and shortly after that, the head accelerates linearly upward reaching the peak at approximately 70ms later. The onset of SCM muscle activation and rearward angular acceleration of the head occur around 110ms; however the muscles usually reaches the peak within 30ms, whereas the backward angular acceleration of the head takes approximately 80ms to reach the maximum value of peak acceleration. The maximum angular displacement of the head is usually observed later than 260ms. Kinematics of head movement and SCM muscle response of subject two upon a fast-unexpected impact is graphically represented in Figure 2.6a and 2.6b.

### 2.3.1 Kinematics of the Torso

Linear torso acceleration: The mean peak magnitude of the linear acceleration for the torso relative to the chair is presented in Table 2.3. The peak acceleration of the torso had a 100% increase in the X axis and a 200% increase in the Y axis with the fast unexpected impact. Difference by gender in the peak acceleration

was not observed in the X-axis (p value 0.571, power 0.086) and Y-axis (p value 0.267, power 0.194). There were no significant differences in the peak acceleration of the torso in the X and Y axes regarding awareness.

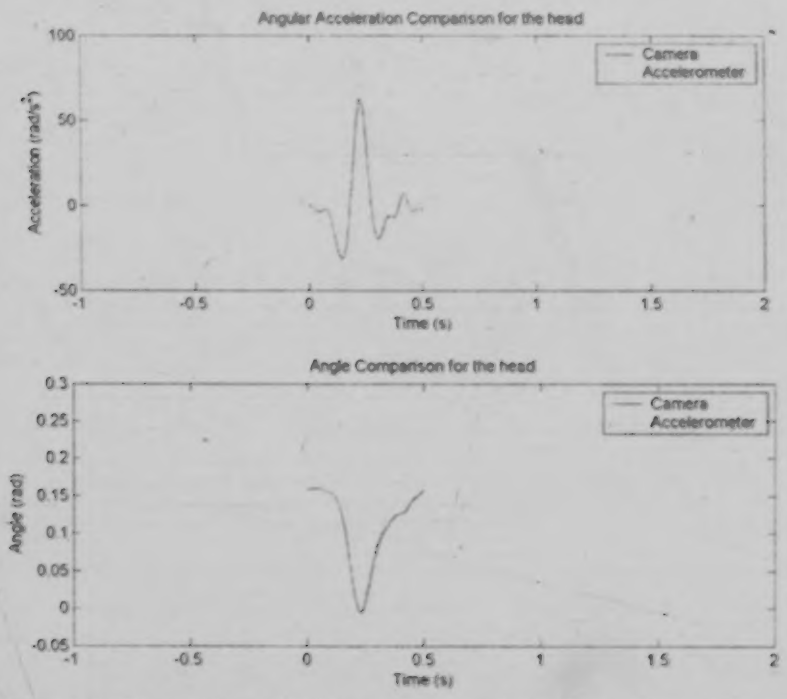


Figure 2.5: Comparison of video camera and accelerometer system recording

No significant differences in the onset time were observed in the rearward torso acceleration in the X axis (p value= 0.100, power 0.147) with increase of impact magnitude. In the X axis, the peak acceleration in the fast unexpected impact reached the peak 10 ms earlier than the peak acceleration of slow impact (p value= 0.014, power =0.789). In the Y-axis, the peak acceleration in the fast unexpected impact reached the peak 49 ms later than the peak in the slow impact (p value <0.016, power 0.845).

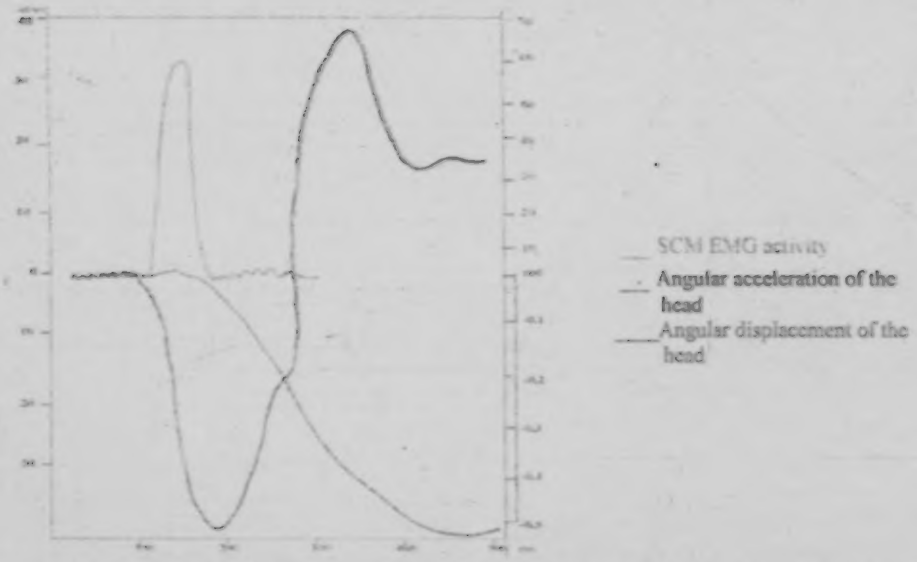


Figure 2.6A: Graphic representation of the SCM EMG activity, head angular acceleration (X and Y axes), and angular head displacement upon impact. Subject 2 upon simulated fast-unexpected impact.

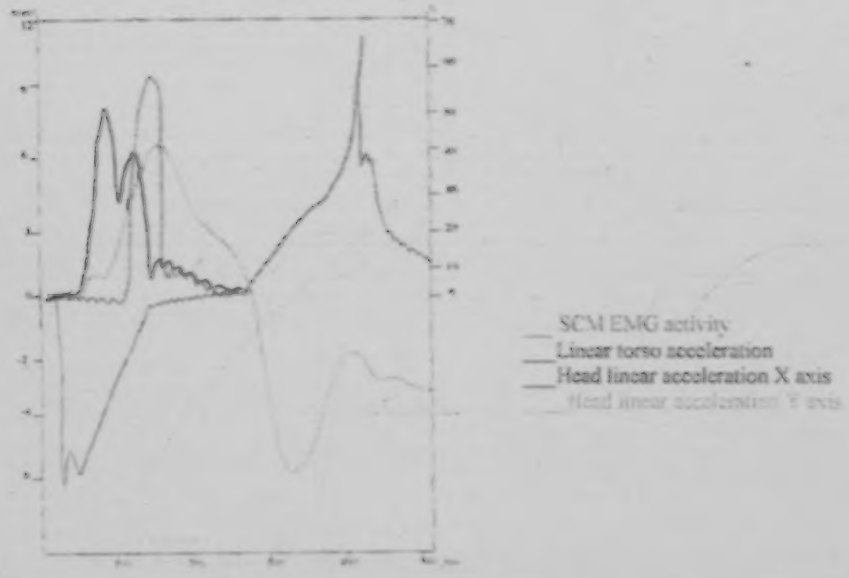


Figure 2.6b: Graphic representation of the SCM EMG activity, head linear acceleration (X and Y axes), and torso acceleration upon impact. Subject 2 upon simulated fast-unexpected impact.

Regarding awareness, there were no significant differences in the onset time of torso acceleration in the X (p value 0.999, power 0.147) and Y (p value = 0.999, power 0.602). Likewise, there were no significant differences in the peak time of torso acceleration in the X ( p value = 0.303, power 0.789) and Y ( p value =0.166, power 0.845) axes) regarding expectation.

### 2.3.2 Kinematics of the Head

Mean peaks of the kinematic events of the head are presented in Table 2.4. Onset and peak times (relative to onset of chair acceleration) of the kinematic events are presented in Table 2.5. Analysis by gender is included; values for females and males are presented separately when a difference by gender was detected.

*Angular head displacement* Rearward angular head displacement increased by over 100% with an increase of the impact magnitude. Gender differences were significant for the low unexpected impacts, but not for the high unexpected and expected impacts. In the slow impact, female subjects presented approximately 100% higher angular displacement than male subjects. Likewise, in the slow impact, female subjects began to rotate their head approximately 95 ms later than male subjects. There was no difference in the magnitude of angular displacement regarding awareness.

*Angular head acceleration:* The peaks of the rearward and forward angular head acceleration increased by 100% and 200% respectively with higher impact magnitude. No significant differences in peak rearward and forward angular head acceleration regarding expectation were detected. Gender differences were identified. In the expected high impact, female subjects presented with 70% higher peak

rearward and 50% higher forward angular head acceleration, compared with male participants. No differences in the onset or peak angular head acceleration times were observed with an increase of impact magnitude.

*Linear head acceleration at the anterior temporal region:* X axis: Rearward and forward linear peak acceleration increased with an increase of impact magnitude by approximately 150% and 260% respectively. No difference in the peaks of the rearward or forward linear accelerations regarding gender was detected. No difference in the peaks of the rearward or forward linear accelerations regarding awareness was detected.

*Linear head acceleration at the anterior temporal region:* Y axis: Upward and downward linear peak acceleration increased by approximately 270% and 320% respectively with the larger impact magnitude. In the fast-unexpected impact, female subjects presented approximately 60% lower upward peak acceleration than male subjects. On the other hand, in the expected impact, female subjects presented with 40% lower downward peak acceleration than male subjects. No difference in the upward or downward peak linear head acceleration regarding awareness was detected.

*Timing of head acceleration:* In the expected fast impact, the rearward peak angular acceleration started approximately 44 ms later than rearward linear acceleration (p value= 0.042, power 0.996). The rearward angular acceleration reached the peak acceleration approximately 76 ms and 84 ms later than the rearward linear acceleration in the slow (p value= 0.001, power 0.977) and in the expected (p value= 0.001, power 0.999) impacts respectively.

### 2.3.3 Muscle Response:

The mean normalized EMG peaks for the SCM muscles for the unexpected and expected impacts are presented in Table 2.6. Onset time and peak time of the EMG peaks for the unexpected and expected impacts are presented in the Table 2.7.

*Normalized SCM muscle EMG:* The magnitude of SCM EMG activity increased with larger impact magnitude. An increase of approximately 128% and 210% in the left and right SCM muscle was observed respectively in the fast-unexpected impact. Gender differences were identified. In the slow impact, SCM EMG activity of female subjects was three times as high as that of male participants. In the fast-unexpected impact, the left SCM EMG peak of female subjects was two times as high as that of male participants. Statistical difference for gender was not identified for the right SCM in the fast unexpected impact, but the variance was high resulting in low statistical power. No differences in onset or peak time of the left and right SCM EMG peaks were detected between the slow and fast unexpected impacts. Significant difference for expectation was not identified for onset, timing of peak EMG and magnitude of EMG response. Statistical power was high for EMG magnitude data, but low for EMG timing data.

There were no differences in magnitude or timing of peak EMG activity regarding expectation in the left and right SCM muscles.

*Temporal relationship between the muscle activity, and head acceleration:* Temporal relationship was assessed in two steps. First, a repeated measures test was used to determine whether the onset time of the EMG peak for the left and right SCM differed. No significant difference was detected (p value 0.999, power 0.999);

therefore, the onset time of both sides were averaged and this new onset data was analyzed with the peak time of the acceleration of the head.

The onset of the SCM peak EMG was approximately 100 ms later than the peak of the linear acceleration in the expected fast (p value 0.001, power 0.999) and unexpected slow (p value 0.0014, power 0.966) impacts. The onset of the SCM EMG activity was approximately 20 ms earlier than the peak of the angular acceleration in the unexpected fast impact (p value 0.013, power 0.746).

## 2.4 Discussion

The current study analyzed the head kinematics and the SCM muscle responses in simulated rear-end impacts. The larger angular acceleration and linear head accelerations observed with an increase in impact magnitude in the present study are in agreement with previous studies[13, 23, 24, 26]. The magnitude of angular displacements reported in the present study are also in agreement with the findings of other studies[18, 21, 24, 27, 20], and do not support the hyperextension theory.[28] The smaller acceleration values reported in the current study compared with previous studies might be due to the lesser impact magnitudes[21, 24].

The role of the SCM muscle in whiplash injury has gained acceptance over the last few years[13-17]. Increased EMG activity was observed at higher impact magnitude, and the results of the current study support the hypothesis that at higher levels of impacts the muscles may become injured[17]. The present study did not identify more rapid SCM muscle response with greater impact velocity. This finding is not in agreement with those of Kumar et al[13] and Brault et al[17]. This disagreement

may be explained by the large variances of the timing variables and the low statistical power (≈0.100) associated with them in the current study.

The relationship between angular and linear acceleration is complex. In the expected fast impacts, onset of angular acceleration occurred on average 44ms later than onset of linear acceleration. Similarly peak angular acceleration was 76-84ms later than peak linear acceleration. Head movement is a combination of translation and rotation. The initial movement was translation followed by combined rotation and translation and ultimately by rotation. The axis of rotation of the head was not calculated and would be an instantaneous rather than fixed axis.

The temporal relationship between the muscle activity and the acceleration of the head is different depending whether the EMG activity was compared with the linear or angular acceleration. If the analysis is made with the linear acceleration, it might be concluded that the muscle activates too late to have an influence on the head kinematics. On the other hand, the same comparison made with the angular acceleration is less definitive, and it supports the concept that the muscle may play a role in the head kinematics upon impact.

The head does not only present a translational but also a rotational movement. The linear acceleration of the head is specific to the location in which it was measured. Its value varies across the head depending on distance from the rotational axis. On the other hand, the value of the angular acceleration of the head is valid for any point of the head. Linear and angular acceleration values of the current study present a significant increase at higher levels of impacts; however, the linear acceleration presents a larger increase, an earlier onset, and reached the peak

acceleration faster than the angular acceleration. Therefore, it might be more appropriate to analyze the cervical muscle response with both types of acceleration rather than assume the linear acceleration as the key movement of the head.

Important gender differences in head kinematics were identified in the present study. Angular peak head acceleration was greater in female subjects. Vertical linear peak head acceleration was lower in females. The findings of Siegmund et al[33] also reported similar gender differences in head accelerations. Kumar et al[23] reported gender difference in rearward linear head acceleration; however, angular head acceleration was also not reported. Greater angular head acceleration in females would increase the risk of cervical injury. Differences in vertical acceleration may be a function of the car seat relationship to the subject's body. Males are generally taller than females and the contact with the back against the car seat in males may result in more upward movement.

Brault et al[17] reported that female subjects activated their cervical muscles at an average of 5% earlier than male subjects. Due to large variances and low statistical power the present study does not allow conclusions regarding gender differences in SCM EMG onset and peak times. The present study did identify gender differences in magnitude of peak EMG with female subjects demonstrating a greater EMG response. This finding is consistent with greater potential muscle injury and higher prevalence of whiplash symptoms reported in females in low impact rear-end collisions. Interpretation of results from previously published studies without differentiation of gender should be viewed with caution.

It has been reported that awareness of an impact and intensity of muscle tension influences the muscle response and the kinematics of the head in a simulated whiplash event.[13, 19, 23, 29, 30] Awareness of an event implies anticipation of such an event.[31] Frank[32] defined two components in such anticipation: temporal and event awareness. The first refers to whether the subject knows about the exact timing in which an event will occur, whereas, the second describes whether the subject knows an event will occur. Recently, Siegmund[31] added a third dimension to the anticipation of an event: amplitude awareness, which refers to the awareness of the amplitude of an imminent event. Magnusson et al[14] did not report significant differences regarding temporal awareness in simulated impacts at 0.5g. Siegmund et al[33] reported that lack of event awareness significantly increased onset and peak latencies of the cervical muscles, and it affected kinematic gender response.

Kumar et al[23] reported that temporal and amplitude awareness of a simulated rear-end impact of 1.4g reduced the peak of the linear acceleration of the head by 30%. Later, the same authors reported that the values of the SCM EMG peaks were significantly lower in the expected impacts.[13] Pope et al[29] reported that training of the subjects prior to impacts had a major influence in the kinematics of the head and muscle response: Pre-impact training consisting of self-bracing manoeuvre produced an increase in the magnitude and duration of muscle activity, decreased duration of the head acceleration, and decreased angular displacement of the head. Similarly, pre-impact tension of the cervical muscles decreases the angular displacement of the head by 30%.[19]

The current findings as well as those of Siegmund et al[33] and Magnusson et al[14] did not report significant differences in muscle responses or head kinematics regarding temporal awareness. Siegmund et al[33] concluded that event awareness, rather than temporal or amplitude awareness, is the component of anticipation event that plays an influential role in the kinematics of the head and neck in a simulated whiplash event. The unexpected events in the present study are similar to the "unalerted" group in the Siegmund et al[33] study. Subjects were aware that they would eventually sustain impact and therefore had event awareness. To remove event awareness requires deception such as used by Siegmund et al.[33] The expected impact events in the present study are similar to the "alerted" group in the Siegmund et al[33] study.

Kumar et al[23] reported that awareness resulted in 30% reduction in peak backward linear head acceleration. Kumar et al[13] used the same methodology to test the role of awareness in cervical muscle EMG. They reported SCM EMG magnitude and EMG latency reduced with expectancy of impact. The two Kumar et al[13,23] studies test expectancy based on the subject being advised of timing and magnitude of impact. There was no record of deception for the unexpected impact. The papers do not state how long the subject was kept blindfolded with loud music prior to the impact. It seems likely however that there was event awareness but not temporal or magnitude awareness. A possible explanation for the differences reported in these studies and the present study may relate to the subjects being informed of the magnitude of impact. The present study utilized only one "alerted" impact magnitude.

It is possible that the subjects did not perceive the simulated impact sufficiently noxious to get injured.

The question of what triggers the muscle to contract remains without an answer. The extent to which the central and peripheral mechanisms play a role in whiplash injury is also debatable. Siegmund et al[16 31] suggested that whiplash injuries resemble reflex muscle responses. They have reported habituation of the cervical muscle response upon multiple whiplash like perturbations[34]. In addition, they stated that the early onset time of the muscle response reported by simulated impact studies suggests a muscle reflex response. However, the current data do not support this theory; the later onset of the muscle activity reported in this study is in agreement with that by Szabo and Welcher[20].

On the other hand, the fact that the muscle response increases with an increase of the magnitude of the impacts, as well as, the decrease of the muscle response onset with an increase of the magnitude of the impact strongly supports a role of a peripheral mechanism. Finally, the controversial results regarding the role of awareness in head kinematics and muscle response add more uncertainty to the neural pathway of whiplash injury.

The muscle reaction generated in a simulated whiplash event affects the whiplash biomechanics and could possibly offer protection or alternatively could cause a mechanical failure. Brault et al[17] reported a lengthening by 3-6% of the SCM upon simulated impacts. This eccentric muscle contraction mechanism is consistent with the delayed onset of muscle symptoms observed in whiplash injuries[35 36]. This mechanism is also in agreement with the generalized muscular hyperalgesia present

in subjects who have experienced whiplash trauma; this generalized central hyperexcitability might be caused through tissue injury[37].

This study presents some limitations. It is possible that the mechanism by which the accelerometer board was attached to the subject has created an additional sensory stimulus that might have altered the muscle response, and head motion. The distance between the head of the participant and the headrest was not recorded, and might account for the gender differences reported in this study. The restriction of the kinematics analysis to the first 750ms after impact did not allow insight regarding angular head displacement in the rebound phase, and its potential relationship with the muscle response. The low magnitude of impacts in this study might have not been sufficient to create an actual simulation of a whiplash event; and therefore, to be perceived sufficiently noxious by the participants in the expected condition. The results of the current study should not be extrapolated to older people since the composition of the muscle tissue is different, and therefore the muscle response might be different.

**2.5 Conclusions**

Based on the findings of the current study, the following can be concluded: There is increased SCM EMG activity and kinematic events of the head with an increased impact magnitude. Linear and angular onset and peak accelerations were not coincident. Gender differences for head kinematics and SCM EMG magnitude were identified. Temporal and amplitude awareness of a simulated impact do not produce differences in the magnitude of muscle response or head kinematic events.

Conclusions regarding gender differences related to timing of EMG response cannot be drawn due to large variances of the variables and low power associated with those findings.

## 2.6 Tables

Table 2.1: Descriptive statistics of demographic data

| | Number | Age (S.D) | Height (S.D) | Weight (S.D) |
|---|---|---|---|---|
| Females | 18 | 25 (3.39) | 169 (8) | 63 (10.83) |
| Males | 23 | 26 (3.19) | 178 (6.91) | 75 (11.99) |

Age is expressed in years, height in centimetres, and weight in kilograms.

Table 2.2: Mean of the linear chair acceleration peaks in the X, Y and Z axes

| | Slow impact (S.D) | Fast impact (S.D) | Slow-fast p value and power |
|---|---|---|---|
| Acceleration X axis $m/s^2$ | 4.48 (.81) | 10.18 (2.20) | 0.001* 0.999 |
| Acceleration Y axis $m/s^2$ | -2.10 (1.47) | -6.10 (4.43) | 0.001* 0.999 |
| Acceleration Z axis $m/s^2$ | 0.36 (.14) | 1.8 (.28) | 0.001* 0.999 |
| Sample | 33 | 68 | |

One way ANOVA . Magnitude of the impacts expressed as mean of the linear acceleration peaks of the sled.

P value and power is presented for each analysis.

* The mean difference is significant at the .05 level.

**Table 2.3: Mean peak of the linear torso acceleration in the X and Y axes.**

| | Sample | Slow Unexpected Impact | Fast Unexpected Impact | Fast Expected Impact | Slow- fast unexpected p value and power | Fast: unexpected-expected p value and Power |
|---|---|---|---|---|---|---|
| Acceleration X axis $m/s^2$ (S.D) | 19M 12 F | 3.36 (.64) | 8.35 (1.35) | 8.27 (1.36) | 0.001* 0.999 | 0.999 0.999 |
| Acceleration Y axis $m/s^2$ (S.D) | 19M 12 F | -2.36 (.57) | -7.07 (1.78) | -7.25 (2.02) | 0.001* 0.999 | 0.999 0.999 |

Repeated measures test. P value and power is presented for each analysis of acceleration. * The mean difference is significant at the .05 level.

**Table 2.4: Mean peaks of the angular displacement, angular acceleration and linear acceleration of the head.**

| Peaks | Units | Slow unexpected Impact (S.D) | Gender P value power | Fast unexpected Impact (S.D) | Gender P value Power | Fast expected Impact (S.D) | Gender P value power | Slow-fast unexpected p value, power | Fast: unexpected-expected p value, power | Sample |
|---|---|---|---|---|---|---|---|---|---|---|
| Rearward angular displacement | Degrees | -4.04 (2.39) M<br>-8.14 (3.69) F<br>-5.86 (3.61 T | .012*<br>.763 | -11.20 (7.71) T | .953<br>.050 | -12.83 (5.41) T | .739<br>.062 | .040*<br>.996 | .999<br>.996 | 10M<br>8 F |
| Rearward ang acc | $Rad/s^2$ | -16.31(4.40) T | .496<br>.101 | -.31.51 (11.23) T | .555<br>.088 | -24.72 (7.14) M<br>-37.44 (12.82) F<br>-30.75 (11.88) T | .015*<br>.724 | .000*<br>.999 | .999<br>.999 | 10 M<br>9 F |
| Forward ang acc | $Rad/s^2$ | 15.10 (6.22) T | .235<br>.214 | 48.45(28.00) T | 205<br>.237 | 33.59 (13.56) M<br>57.19 (36.32) F<br>44.85 (23.65) T | .024*<br>.647 | .000*<br>.999 | .999<br>.999 | 10 M<br>9 F |
| Rearward li acc X axis | $M/s^2$ | -5.04 (2.24) T | .243<br>.210 | -12.95 (7.51) T | .174<br>.269 | -12.54 (7.41) T | .330<br>.159 | .000*<br>.999 | .999<br>.999 | 17 M<br>8 F |
| Forward li acc X axis | $M/s^2$ | 2.18 (.83) T | .739<br>.062 | 7.95 (4.28) T | .636<br>.075 | 5.91(2.28) T | .378<br>.138 | .000*<br>.999 | .102<br>.999 | 17 M<br>8F |
| Upward li acc Y axis | $M/s^2$ | 2.82 (3.44) T | .440<br>.118 | 9.91 (9.16) M<br>3.69 (1.83) F<br>7.69 (7.95) T | .045*<br>.527 | 5.46 (4.68) T | .604<br>.080 | .005*<br>.930 | .999<br>.930 | 18 M<br>10 F |
| Downward li acc Y axis | $M/s^2$ | -2.65 (2.72) T | .530<br>.094 | -8.44 (7.19) T | .549<br>.090 | -4.69 (2.16) M<br>-7.94 (4.71) F<br>-5.85 (3.59) T | .019*<br>.677 | .001*<br>.999 | .659<br>.999 | 18 M<br>10 F |

Repeated measures test. M refers to male and F to females. T refers to the average of females and males. P value and power is presented for each analysis. * The mean difference is significant at the .05 level.
Rearward ang acc refers to the rearward angular acceleration. Forward ang acc refers to the forward angular acceleration of the head.
Upward li acc refers to the upward linear acceleration. Downward li acc refers to the downward linear acceleration.

Table 2.5: Mean of the onset and peak times of the angular displacement, angular acceleration and linear acceleration of the head.

| | Slow unexpected Impact (S.D) | Gender P value Power | Fast unexpected Impact (S.D) | Fast expected Impact (S.D) | Slow-fast unexpected p value, Power | Fast: unexpected-expected p value, Power | Sample |
|---|---|---|---|---|---|---|---|
| Onset time rearward angular displ | 100.7 (63.58) T | | 128.21 (69.88) T | 124.79 (54.671) T | 0.734<br>0.160 | 0.999<br>0.160 | 8 M<br>6 F |
| Peak time Rearward angular displ | 214.38 (91.10) M<br>310.83 (19.26) F<br>255.714 (84.05) T | 0.027<br>0.641 | 267.50 (108.49) T | 276.57 (63.18) T | 0.999<br>0.069 | 0.999<br>0.069 | 8 M<br>6 F |
| Onset time Rearward ang acc | 35.89 (18.59) T | | 59.28 (73.39) T | 61.13 (49.34) T | 0.620<br>0.408 | 0.999<br>0.408 | 9M<br>7 F |
| Peak time Rearward ang acc | 108.72 (23.19) T | | 140.4 (74.92) T | 123.68 (32.27) T | 0.227<br>0.678 | 0.981<br>0.678 | 9M<br>7 F |
| Onset time Forward ang acc | 221.95 (93.47) T | | 210.98 (76.65)T | 205.65 (61.5) T | 0.999<br>0.080 | 0.999<br>0.080 | 9M<br>7 F |
| Peak time Forward ang acc | 269.96 (92.78) T | | 268.68 (74.14) T | 259.57 (60.76) T | 0.999<br>0.067 | 0.999<br>0.067 | 9M<br>7 F |
| Onset time Rearward li acc | 21.2 (58.96) T | | 52.91 (97.20) T | 17.65 (59.35) T | 0.495<br>0.215 | 0.456<br>0.215 | 15 M<br>8 F |
| Peak time Rearward li acc | 32.70 (63.54) T | | 75.00 (112.41) T | 29.43 (71.51) T | 0.240<br>0.301 | 0.333<br>0.301 | 15 M<br>8 F |
| Onset time Forward li acc | 290.30 (173.15) T | | 301.39 (183.11) T | 312.78 (139.42) T | 0.999<br>0.064 | 0.999<br>0.064 | 15 M<br>8 F |
| Peak time Forward li acc | 300.09 (166.05) T | | 322.04 (165.93) T | 328.96 (130.56) T | 0.999<br>0.079 | 0.999<br>0.079 | 15 M<br>8 F |

Repeated measures test. Time is expressed in milliseconds. M refers to male and F to females. T refers to the average of females and males.
P value and power is presented for each analysis. * The mean difference is significant at the .05 level.
Rearward ang acc refers to the rearward angular acceleration. Forward ang acc refers to the forward angular acceleration of the head.

Table 2.6: Mean of the electromyogram peaks of the right and left SCM muscles

| EMG SCM | Unit | Slow unexpected impact (S.D) | Gender P value power | Fast unexpected Impact (S.D) | Gender P value power | Fast expected impact (S.D) | Gender p value power | Slow-fast unexpected p value, power | Fast: unexpected-expected p value, power | Sample |
|---|---|---|---|---|---|---|---|---|---|---|
| Left | % | 15.16 (15.10) M<br>56.94 (64.12) F<br>32.83 (47.06) T | 0.043*<br>0.536 | 55.84 (31.54) M<br>101.31 (120.11) F<br>75.07 (82.78) T | 0.038*<br>0.559 | 51.23 (36.51) M<br>123.96 (120.52) F<br>82.00 (88.88) T | 0.093<br>0.390 | 0.001*<br>0.997 | 0.563<br>0.997 | 15 M<br>11 F |
| Right | % | 19.99 (16.40) M<br>55.13 (51.13) F<br>34.53 (36.61) T | 0.011*<br>0.747 | 85.10 (36.56) M<br>141.72 (136.17) F<br>107.34 (91.94) T | 0.099<br>0.377 | 85.11(53.04) M<br>132.55 (95.18) F<br>104.74 (75.71) | 0.142<br>0.308 | 0.001*<br>0.999 | 0.999<br>0.999 | 17 M<br>12 F |

Repeated measures test. Normalized EMG is expressed in percentage. M refers to male and F to females. T refers to the average of females and males.
P value and power is presented for each time analysis. * The mean difference is significant at the .05 level.

Table 2.7: Mean of the onset and peak times of the electromyogram peaks of the right and left SCM muscles

| | Slow unexpected impact (S.D) | Fast unexpected Impact (S.D) | Fast expected impact (S.D) | Slow-fast unexpected p value, power | Fast: unexpected-expected p value, power | Sample |
|---|---|---|---|---|---|---|
| Onset time Left SCM | 116.46 (106.80) T | 107.00 (49.55) T | 100.04 (54.74) T | 0.999<br>0.111 | 0.999<br>0.111 | 15 M<br>11 F |
| Onset time Right SCM | 142.21 (128.58) T | 142.4 (125.02) T | 119.45 (106.13) T | 0.999<br>0.117 | 0.999<br>0.117 | 17 M<br>12 F |
| Peak time Left SCM | 132.50 (108.21) T | 128.6 (39.07) T | 124.19 (46.14) T | 0.999<br>0.070 | 0.999<br>0.070 | 15 M<br>11 F |
| Peak time Right SCM | 159.6 (123.45) T | 160.10 (120.67) T | -141.90 (101.08) T | 0.999<br>0.100 | 0.999<br>0.100 | 17 M<br>12 F |

Repeated measures test. Time is expressed in milliseconds. M refers to male and F to females.
T refers to the average of females and males.
P value and power is presented for each time analysis. * The mean difference is significant at the .05 level.

**2.7 Reference List**


1. Otremski I, Marsh JL, Wilde BR, McLardy Smith PD, Newman RJ. Soft tissue cervical spinal injuries in motor vehicle accidents. Injury 1989; 20(6):349-51.

2. Malleson A. Chronic whiplash syndrome. Psychosocial epidemic. Canadian Family Physician 1994; 40:1906-9.

3. Yoganandan N, Pintar FA, Kleinberger M. Whiplash injury. Biomechanical experimentation. Spine 1999; 24(1):83-5.

4. Spitzer WO, Skovron ML, Salmi LR *et al.* Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. Spine 1995; 20(8 Suppl):1S-73S.

5. Bogduk N, Marsland A. The cervical zygapophysial joints as a source of neck pain. Spine 1988; 13(6):610-7.

6. Aprill C, Bogduk N. The prevalence of cervical zygapophyseal joint pain: A first approximation. Spine 1992; 17(7):744-7.

7. Barnsley L, Lord S, Wallis B, Bogduk N. False-positive rates of cervical zygapophysial joint blocks. Clin J Pain 1993; 9(2):124-30.

8. Winkelstein B and Myers B. Cervical motion segment, combined loading, muscle forces, and facet joint: A mechanical hypothesis for whiplash injury. In: Yoganandan N, Pintar F, Editors. Frontiers in whiplash trauma: clinical and biomechanical. Washington DC USA: IOS PRESS; 2000: 249-62.

9. Winkelstein BA, Nightingale RW, Richardson WJ, Myers BS. The cervical facet

capsule and its role in whiplash injury: a biomechanical investigation. Spine 2000; 25(10):1238-46.

10. Winkelstein BA, McLendon RE, Barbir A, Myers BS. An anatomical investigation of the human cervical facet capsule, quantifying muscle insertion area. J Anat 2001; 198(Pt 4):455-61.

11. Siegmund GP, Myers BS, Davis MB, Bohnet HF, Winkelstein BA. Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading. Spine 2001; 26(19):2095-101.

12. Panjabi MM, Cholewicki J, Nibu K, Grauer J, Vahldiek M. Capsular ligament stretches during in vitro whiplash simulations. J Spinal Disord 1998; 11(3):227-32.

13. Kumar S, Narayan Y, Amell T. An electromyographic study of low-velocity rear-end impacts. Spine 2002; 27(10):1044-55.

14. Magnusson ML, Pope MH, Hasselquist L *et al*. Cervical electromyographic activity during low-speed rear impact. Eur Spine J 1999; 8(2):118-25.

15. Siegmund GP, Brault JR. Role of cervical muscles during whiplash. In: Yoganadan N, Pintar FA, Editors. Frontiers in whiplash trauma: clinical and biomechanical. Washington, DC USA: IOS Press; 2000: 295-320.

16. Siegmund G, Brault J, Chimich D. Do cervical muscles play a role in whiplash injury? Journal of Whiplash and Related Disorders 2002; 1:23-40.

17. Brault JR, Siegmund GP, Wheeler JB. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. Clin Biomech (Bristol, Avon)

2000; 15(6):426-35.

18. Castro WH, Schilgen M, Meyer S, Weber M, Peuker C, Wortler K. Do "whiplash injuries" occur in low-speed rear impacts? Eur Spine J 1997; 6(6):366-75.

19. Ono K, Kaneoka K, Wittek A, Kazjer J. Paper 973340 Cervical Injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Annual Stapp Car Crash Conference . Warrendale , Pennsylvania, USA: Society of Automotive Engineers, Inc., 1997: 339-56.

20. Szabo T, Welcher J. Paper 962432 Human subject kinematics and electromyographic activity during low speed rear impacts. 40th Stapp Car Crash Conference. Warrendale, Pennsylvania, USA: Society of Automotive Engineers Inc. 1996: 295-314.

21. McConnell W, Howard RP, Poppel JV *et al.* Paper 952724 Human head and neck kinematics after low velocity rear-end impactsUnderstanding "whiplash". 39th Stapp Car Crash Conference . Warrendale, Pennsylvania: Society Automotive Engineers, Inc, 1995: 3106-29.

22. Sturzenegger M, DiStefano G, Radanov BP, Schnidrig A. Presenting symptoms and signs after whiplash injury: the influence of accident mechanisms. Neurology 1994; 44(4):688-93.

23. Kumar S, Narayan Y, Amell T. Role of awareness in head-neck acceleration in low velocity rear-end impacts. Accid Anal Prev 2000; 32(2):233-41.

24. Siegmund G, King D, Lawrence J, Wheeler J, Brault J and Smith T. Paper 973341

Head/neck kinematic response of human subjects in low-speed rear-end collisions. 41st Annual Stapp Car Crash Conference. Warrendale, Pennsylvania, USA : Society Automotive Engineers, Inc, 1997: p 357-85.

25. Basmajian J, de Luca C. Description and Analysis of teh EMG signal. In. Muscles Alive: Their functions revealed by Electromyography. Fifth Edition edition. Baltimore, USA: Williams and Wilkins , 1985: 65-100.

26. Welcher J, Szabo T, Voss D. 2001-01-0899 Human Occupant Motion in Rear-end Impacts: Effects of incremental increases in velocity change. SAE 2001 World Congress . Warrendale, Pennsylvania: Society of Automotive, Inc, 2001.

27. McConnell W, Howard RP, Guzman HM *et al*. Paper 930889 Analysis of human test subject kinematic responses to low velocity rear-end impacts. Presentation to . SAE International Congress and Exposition. Warrendale, Pennsylvania, USA: Society of Automotive Engineers, Inc , 1993: 21-30.

28. Weinberg S, Lapointe H. Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 1987; 45(8):653-6.

29. Pope M, Magnusson, M *et al*. Electromyographic changes under whiplash loading. in: Yoganadan N et al, Editors. Frontiers in Head and Neck Trauma. IOS Press, 1998: 338-43.

30. Ono K, Kanno M. Influences of the physical parameters on the risk to neck injuries in low impact speed rear-end collisions. Accid Anal Prev 1996; 28(4):493-9.

31. Siegmund G. The reflex response of human neck muscles to whiplash-like

perturbations [Doctoral Thesis]. Vancouver, British Columbia, Canada: University of British Columbia, 2001.

32. Frank J. S. Spinal motor preparation in humans . Electroencephalography and Clinical Neurophysiology 1986; 63:361-70.

33. Siegmund GP, Sanderson DJ, Myers BS, Inglis JT. Awareness affects the response of human subjects exposed to a single whiplash-like perturbation. Spine 2003; 28(7):671-9.

34. Siegmund GP, Sanderson DJ, Myers BS, Inglis T. Rapid neck muscle adaptation alters the head kinematics of aware and unaware subjects undergoing multiple whiplash-like perturbations. Journal of Biomechanics 2003; 36:473-82.

35. McCully K and Faulkner J. Injury to skeletal muscle fibers of mice following lengthening contractions. Journal of Applied Physiology 1985; 59(1):119-26.

36. Newham D.J. The consequences of eccentric contractions and their relationship to delayed onset muscle pain. European Journal of Applied Physiology 1988; 57:353-9.

37. Koelbaek Johansen M, Graven-Nielsen T, Olesen A , Arendt-Nielsen L. Generalised muscular hyperalgesia in chronic whiplash syndrome. Pain 1999; 83:229-34.

# Chapter Three

## Discussion

## and

## Recommendations

## 3.1 General Discussion

The biomechanical mechanism of whiplash injury is an ongoing topic of debate. The many variables involved in this mechanism, the ethical limitations to reproduce impacts of a similar magnitude as may occur in a real collision, and the wide range of methodologies found in the scientific literature add complexity to its understanding.

Whiplash injuries are a major health problem and they have significant economic consequences in most industrialized countries[1-3]. In whiplash injuries, cervical and masticatory muscle function as well as the temporomandibular joints may be involved[4-10], but the severity of the whiplash trauma often does not correlate with the seriousness of the clinical symptoms[11]. Scientific findings have suggested that damage to the soft tissue of the cervical spine might be responsible for the neck pain; structures such as the facet joint[12-14], capsule and ligaments[15 16 17-20] are a few examples.

Recently, the role of the cervical muscle in whiplash injury has gained acceptance[21-23]. It has been reported that the cervical muscle response influences the kinematic events of the head[24]. Research findings have revealed that pre-impact muscle tension decreases the magnitude of head motion[25 26]. Controversial results have been reported regarding the effect of awareness in muscle response and kinematic events of the head[24 27-29]. It has been suggested that this disagreement might be due to the use of a plastic chair rather than a car seat in the experimental setting[30]. Differences by gender in muscle response and kinematic events of the head have also

been contradictory. It has been suggested that the sample size might be responsible for such discrepancy[31].

The current study was designed to analyze the cervical and masticatory muscle response, and the kinematic events of the head and mandible upon progressive accelerations. However, the scope of the current thesis is restricted to the analysis of the kinematics of the head and SCM muscle response.

This study was performed with the use of a car seat and surface EMG. The participants were exposed to three impacts: three unexpected impacts of a slow, medium and fast magnitude, and one expected impact. The expected impact was the same magnitude as the fast-unexpected impact. Statistical analysis revealed that the unexpected impacts were significantly different. However, the difference between the slow and medium impact was unperceivable to the investigators. Therefore, it is unlikely that this statistically significant finding fulfills the original goal of doing three unexpected impacts of progressive accelerations. In addition, further statistical analysis revealed no significant difference of the head kinematic variables or muscle response between the slow and medium impacts, although the variables always presented higher values in the medium impact. As a result of this preliminary analysis, a new statistical analysis was done excluding the medium impact.

It was assumed that the motion in the medio-lateral plane would be meaningless; therefore, the analysis of the head kinematics was restricted to the X and Y-axes. This assumption was confirmed with statistical analysis that revealed that the movement in the lateral plane was minimum.

Kinematics of head movement was simultaneously recorded with a video camera and the custom designed accelerometer system. The recordings of the head kinematics from two sources allowed verification of the quality of the data. A consistent reading between the two systems reported in this study ensured that these data were reliable. Positional and acceleration data were obtained from the video camera and acceleration system respectively; this way of processing the raw files minimized the introduction of mathematical error.

The results of this study revealed an increase of the electromyogram and head kinematic peaks with an increase in impact magnitude. The magnitude of angular displacements of the head are in agreement with other studies[2 31-34] and do not support the hyperextension theory[35]. The findings of the current study revealed that temporal and amplitude awareness did not present an effect in the muscle response or kinematic events of the head. Female subjects presented higher kinematic events than male subjects in some expected and unexpected impacts. They also presented higher SCM EMG activity than male subjects in most of the unexpected impacts.

Brault et al[22] reported that female subjects presented an earlier onset than male subjects of the SCM muscle EMG activity. Siegmund et al[29] reported that event awareness influences gender head kinematic response. As Siegmund et al[31] suggested, the greater sample size of these and the current study might allow the detection of such differences. However, it remains uncertain to which extent this gender difference is due to only physical differences in size and weight.

The findings of the present study is in agreement with other studies regarding the lack of influence of temporal and amplitude awareness in muscle response or

kinematic events[28 30]. It is difficult to simulate the actual conditions in a lab setting; participants are aware in advance of the magnitude of the impacts and the risks of experiencing symptoms due to impacts. Therefore, subjects might not perceive the simulated impacts sufficiently noxious to get injured. This might explain the lack of significant low muscle response and head acceleration when subjects wee exposed to the expected impact. Recently, Siegmund et al[29] reported that event awareness rather than temporal or amplitude awareness influences head kinematics.

The analysis of the temporal relationship between muscle response and kinematic events of the head may be seen from different views. Panjabi et al[36] reported a bi-phasic kinematic response of the cervical spine upon simulated whiplash loads. The spine forms an S shape curve with flexion at the upper levels and hyperextension at the lower levels. In the second phase, all levels of the cervical spine are extended and the head reached its maximum extension. This S-shaped formation is in agreement with others in vivo and in vitro studies[37 38]. It has been reported that this S shape formation occurs approximately 75ms after impact[36], and it is this onset time in conjunction with the belief that the muscles need another 100ms to reach their maximum contraction activity that has supported the concept that muscles activate too late to influence head kinematics[39].

In the current study, the temporal relationship between muscle activity and angular acceleration of the head revealed that the SCM muscle activated before the peak of angular acceleration reached its maximum value. The increase of the SCM EMG activity over the maximum voluntary contraction in the fast impacts suggests that the severity of muscle damage increases with the magnitude of the impact. This

is in agreement with findings of Kumar et al[24] and Brault et al[22] In addition, the lengthening muscle contraction of the SCM muscle in simulated impacts has been reported[22]. These findings support the muscle strain theory[40] and might be responsible of the delayed onset muscle soreness present in subjects who have experienced whiplash trauma.

## 3.2 Limitations

In the current study, the accelerometer board was attached to the subject through a dental tray. This might have created an additional sensory stimulus that might have altered the muscle response and the kinematics of the head. McConnell et al[33] also used a bite block to attach accelerometers that measure the kinematic events of the head. The onset time of the rearward angular displacement reported by that study is in agreement with the current study, but the peak time in that study is earlier than in the current study.

The restriction of the kinematics analysis to the first 750ms after impact did not allow an insight into the angular displacement in the rebound phase and its potential relationship with the muscle response or kinematic events. McConnell et al[33] reported that the subjects almost achieved their pre-impact position within the first 600ms. The longer time taken to reach the peak kinematic events in the current study might be due to the lower magnitude of the impacts, seat back and/or head restraint properties.

The distance between the head of the participant and the headrest was not recorded, and might be accounted for the gender differences reported in this study. It

was also impossible to determine if there was any contact between the subject's heads and head restraint. Recording of these data would have been allowed to relate head restraint distance with kinematics of the head and muscle response. Siegmund et al[41] reported that larger horizontal distances between the head of the subject and head restraint allowed larger variance in head motion prior to its contact with the head restraint.

As was previously discussed, the participants might not have perceived the simulated impacts to be sufficiently noxious; therefore, the goal of producing lack of temporal and amplitude awareness might not have been fully achieved.

The results of the current study should not be extrapolated to older people since the composition of the muscle tissue is different and, therefore, the muscle response might be different.

Forty participants completed the experimental phase of the current study. However, technical problems resulted in missing data for up to 50% of the original samples in some variables. This decrease in the sample size might be accounted for the low power associated with the detection of differences in onset and peak time of all the variables.

## 3.3 Recommendations for Future Investigations

A future study may consider analyzing muscle response and human kinematic events upon progressive accelerations. However, this goal might be difficult to achieve due to ethical restrictions and to the sensitivity of the sled system.

Future studies should consider not only the measurement of the height of the subject but also, a measurement of the vertical and horizontal distance between the head of the subject and head restraint. This will allow assessment of its influence in the kinematic events of the head.

For a future study, it would be appropriate to have only one recording system. If two different recording systems were used, it would be appropriate to do preliminary tests to assess their sensitivity and recording capability.

Future research projects in this field should include a pilot study, in which a preliminary processing of the raw data is made. This may give a better understanding of the type of technical problems and, therefore, how they may be minimized or solved. It may also give an idea of how much the sample size might be increased to compensate the possible missing data.

A future research project may consider the measurement of the angular acceleration of the torso. This will provide a better understanding of its kinematics and its relationship with the head kinematics. In addition, the placement of a marker in the sternum and in the chair will facilitate obtaining linear displacement of the torso relative to the chair and linear displacement of the head relative to the torso. It has been suggested that kinematic values related to the torso might give a better understanding of human head neck kinematics upon impacts[31 39].

### 3.4 References


1. Malleson A. Chronic whiplash syndrome. Psychosocial epidemic. Canadian Family Physician 1994; 40:1906-9.

2. Castro WH, Schilgen M, Meyer S, Weber M, Peuker C, Wortler K. Do "whiplash injuries" occur in low-speed rear impacts? Eur Spine J 1997; 6(6):366-75.

3. Spitzer WO, Skovron ML, Salmi LR *et al.* Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. Spine 1995; 20(8 Suppl):1S-73S.

4. Burgess JA, Kolbinson DA, Lee PT, Epstein JB. Motor vehicle accidents and TMDs: assessing the relationship. J Am Dent Assoc 1996; 127(12):1767-72.

5. Friedman MH, Weisberg J. The craniocervical connection: a retrospective analysis of 300 whiplash patients with cervical and temporomandibular disorders. Cranio 2000; 18(3):163-7.

6. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: initial signs, symptoms, and diagnostic characteristics. J Orofac Pain 1997; 11(3):206-14.

7. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. A comparison of TMD patients with or without prior motor vehicle accident involvement: treatment and outcomes. J Orofac Pain 1997; 11(4):337-45.

8. Kolbinson DA, Epstein JB, Senthilselvan A, Burgess JA. Effect of impact and injury characteristics on post-motor vehicle accident temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998; 85(6):665-73.

9. Maimaris C, Barnes MR, Allen MJ. 'Whiplash injuries' of the neck: a retrospective study. Injury 1988; 19(6):393-6.

10. Versteegen GJ, Kingma J, Meijler WJ, ten Duis HJ. Neck sprain after motor vehicle accidents in drivers and passengers. Eur Spine J 2000; 9(6):547-52.

11. Panjabi MM, Nibu K, Cholewicki J. Whiplash injuries and the potential for mechanical instability. Eur Spine J 1998; 7(6):484-92.

12. Bogduk N, Marsland A. The cervical zygapophysial joints as a source of neck pain. Spine 1988; 13(6):610-7.

13. Aprill C, Bogduk N. The prevalence of cervical zygapophyseal joint pain: A first approximation. Spine 1992; 17(7):744-7.

14. Barnsley L, Lord S, Wallis B, Bogduk N. False-positive rates of cervical zygapophysial joint blocks. Clin J Pain 1993; 9(2):124-30.

15. Grauer J, Panjabi MM, Cholewicki J, Nibu K, Dvorak J. Whiplash produces an S-shaped curvature of the neck with hyperextension at lower levels. Spine 1997; 22(21):2489-92.

16. Panjabi MM, Cholewicki J, Nibu K, Grauer J, Vahldiek M. Capsular ligament stretches during in vitro whiplash simulations. J Spinal Disord 1998; 11(3):227-32.

17. Winkelstein B and Myers B. Cervical motion segment, combined loading, muscle forces, and facet joint: A mechanical hypothesis for whiplash injury. In: Yoganandan N, Pintar F, Editors. Frontiers in whiplash trauma: clinical and biomechanical. Washington DC USA: IOS PRESS; 2000: 249-62.

18. Winkelstein BA, Nightingale RW, Richardson WJ, Myers BS. The cervical facet capsule and its role in whiplash injury: a biomechanical investigation. Spine 2000; 25(10):1238-46.

19. Winkelstein BA, McLendon RE, Barbir A, Myers BS. An anatomical investigation of the human cervical facet capsule, quantifying muscle insertion area. J Anat 2001; 198(Pt 4):455-61.

20. Siegmund GP, Myers BS, Davis MB, Bohnet HF, Winkelstein BA. Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading. Spine 2001; 26(19):2095-101.

21. Siegmund GP, Brault JR. Role of cervical muscles during whiplash. In: Yoganadan N, Pintar FA, Editors. Frontiers in whiplash trauma: clinical and biomechanical. Washington, DC USA: IOS Press; 2000: 295-320.

22. Brault JR, Siegmund GP, Wheeler JB. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. Clin Biomech (Bristol, Avon) 2000; 15(6):426-35.

23. Siegmund G, Brault J, Chimich D. Do cervical muscles play a role in whiplash injury? Journal of Whiplash and Related Disorders 2002; 1:23-40.

24. Kumar S, Narayan Y, Amell T. An electromyographic study of low-velocity rear-end impacts. Spine 2002; 27(10):1044-55.

25. Pope M, Magnusson, M *et al.* Electromyographic changes under whiplash loading. in: Yoganadan N et al, Editors. Frontiers in Head and Neck Trauma. IOS Press, 1998: 338-43.

26. Ono K, Kaneoka K, Wittek A, Kazjer J. Paper 973340 Cervical Injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Annual Stapp Car Crash Conference . Warrendale , Pennsylvania, USA: Society of Automotive Engineers, Inc., 1997: 339-56.

27. Kumar S, Narayan Y, Amell T. Role of awareness in head-neck acceleration in low velocity rear-end impacts. Accid Anal Prev 2000; 32(2):233-41.

28. Magnusson ML, Pope MH, Hasselquist L *et al.* Cervical electromyographic activity during low-speed rear impact. Eur Spine J 1999; 8(2):118-25.

29. Siegmund GP, Sanderson DJ, Myers BS, Inglis JT. Awareness affects the response of human subjects exposed to a single whiplash-like perturbation. Spine 2003; 28(7):671-9.

30. Siegmund G. The reflex response of human neck muscles to whiplash-like perturbations [Doctoral Thesis]. Vancouver, British Columbia, Canada: University of British Columbia, 2001.

31. Siegmund G, King D, Lawrence J, Wheeler J, Brault J and Smith T. Paper 973341 Head/neck kinematic response of human subjects in low-speed rear-end collisions. 41st Annual Stapp Car Crash Conference. Warrendale, Pennsylvania, USA : Society Automotive Engineers, Inc, 1997: p 357-85.

32. McConnell W, Howard RP, Guzman HM *et al.* Paper 930889 Analysis of human test subject kinematic responses to low velocity rear-end impacts. Presentation to . SAE International Congress and Exposition. Warrendale, Pennsylvania, USA: Society of Automotive Engineers, Inc , 1993: 21-30.

33. McConnell W, Howard RP, Poppel JV *et al.* Paper 952724 Human head and neck kinematics after low velocity rear-end impactsUnderstanding "whiplash". 39th Stapp Car Crash Conference . Warrendale, Pennsylvania: Society Automotive Engineers, Inc, 1995: 3106-29.

34. Szabo T, Welcher J. Paper 962432 Human subject kinematics and electromyographic activity during low speed rear impacts. 40th Stapp Car Crash Conference. Warrendale, Pennsylvania, USA: Society of Automotive Engineers Inc. 1996: 295-314.

35. Weinberg S, Lapointe H. Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 1987; 45(8):653-6.

36. Panjabi MM, Cholewicki J, Nibu K, Grauer JN, Babat LB, Dvorak J. Mechanism of whiplash injury. Clin Biomech (Bristol, Avon) 1998; 13(4-5):239-49.

37. Kaneoka K, Ono K, Inami S, Hayashi K. Motion analysis of cervical vertebrae during whiplash loading. Spine 1999; 24(8):763-9; discussion 770.

38. Cusick J, Pintar F, Yoganandan N. Whiplash Syndrome: Kinematic factors influenccing pain patterns. Spine 2001; 26(11):1252-8.

39. Cholewicki J, Panjabi MM, Nibu K, Babat LB, Grauer JN, Dvorak J. Head kinematics during in vitro whiplash simulation. Accid Anal Prev 1998; 30(4):469-79.

40. Szabo T. Influence of Seat Properties on Occupant Kinematics and Injury Potential in

Low-Speed Rear Impacts. In : Yoganandan N, Pintar FA , Editors. Frontiers in Whiplash: clinical and biomechanical. Washington DC, USA: IOS Press, 2000: 348-71.

41. Siegmund GP, Heinrichs BE, Wheeler JB. The influence of head restraint and occupant factors on peak head/neck kinematics in low-speed rear-end collisions. Accid Anal Prev 1999; 31(4):393-407.

# Chapter Four

## Appendix

## 4.1 Ethics Approval

Health Research Ethics Board biomedical research health research
2J2.27 Walter Mackenzie Centre, University of Alberta, Edmonton, Alberta T6G 2R7 | 3-48 Corbett Hall, University of Alberta, Edmonton, Alberta T6G 2G4

*UNIVERSITY OF ALBERTA HEALTH SCIENCES FACULTIES,*
*CAPITAL HEALTH AUTHORITY, AND CARITAS HEALTH GROUP*

**HEALTH RESEARCH ETHICS APPROVAL**

| | |
|---|---|
| **Date:** | August 2002 |
| **Name of Applicant:** | Dr Ivonne Hernandez |
| **Organization:** | University of Alberta |
| **Department:** | Dentistry |
| **Project Title:** | An electromyographic study of the masseter and sternocleidomastoid muscles in stimulated low velocity near-end impacts |

The Health Research Ethics Board (HREB) has reviewed the protocol for this project and found it to be acceptable within the limitations of human experimentation. The HREB has also reviewed and approved the subject information letter and consent form.

The deliberations of the HREB included all elements described in Section 50 of the *Health Information Act*, and found the study to be in compliance with all the applicable requirements of the Act. The HREB determined that consent be obtained for the disclosure of the health information to be used in the research from the individuals who are the subjects of the information.

The approval for the study as presented is valid for one year. It may be extended following completion of the yearly report form. Any proposed changes to the study must be submitted to the Health Research Ethics Board for approval. Written notification must be sent to the HREB when the project is complete or terminated.

Dr. Sharon Warfen
Chair of the Health Research Ethics Board (B: Health Research)

File number: B-060602-DENT

University of Alberta | Capital Health | CARITAS HEALTH GROUP